PROSPECTIVE ANALYSIS OF FACTORS DECIDING SURGICAL INTERVENTION IN LIVER ABSCESS

conducted at

COIMBATORE MEDICAL COLLEGE

Submitted in partial fulfillment of the requirements

for the award of the degree

M.S. GENERAL SURGERY

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

MAY 2019

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

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Name of the Candidate:		**Dr. Mohammed Sanoob .M .K**
Course	:	**MS (General Surgery) Post Graduate**
Period of Study	:	**1 year**
College	:	**Coimbatore Medical College & Hospital.**
Dissertation Topic	:	**Prospective analysis of factors deciding surgical intervention in liver abscess.**

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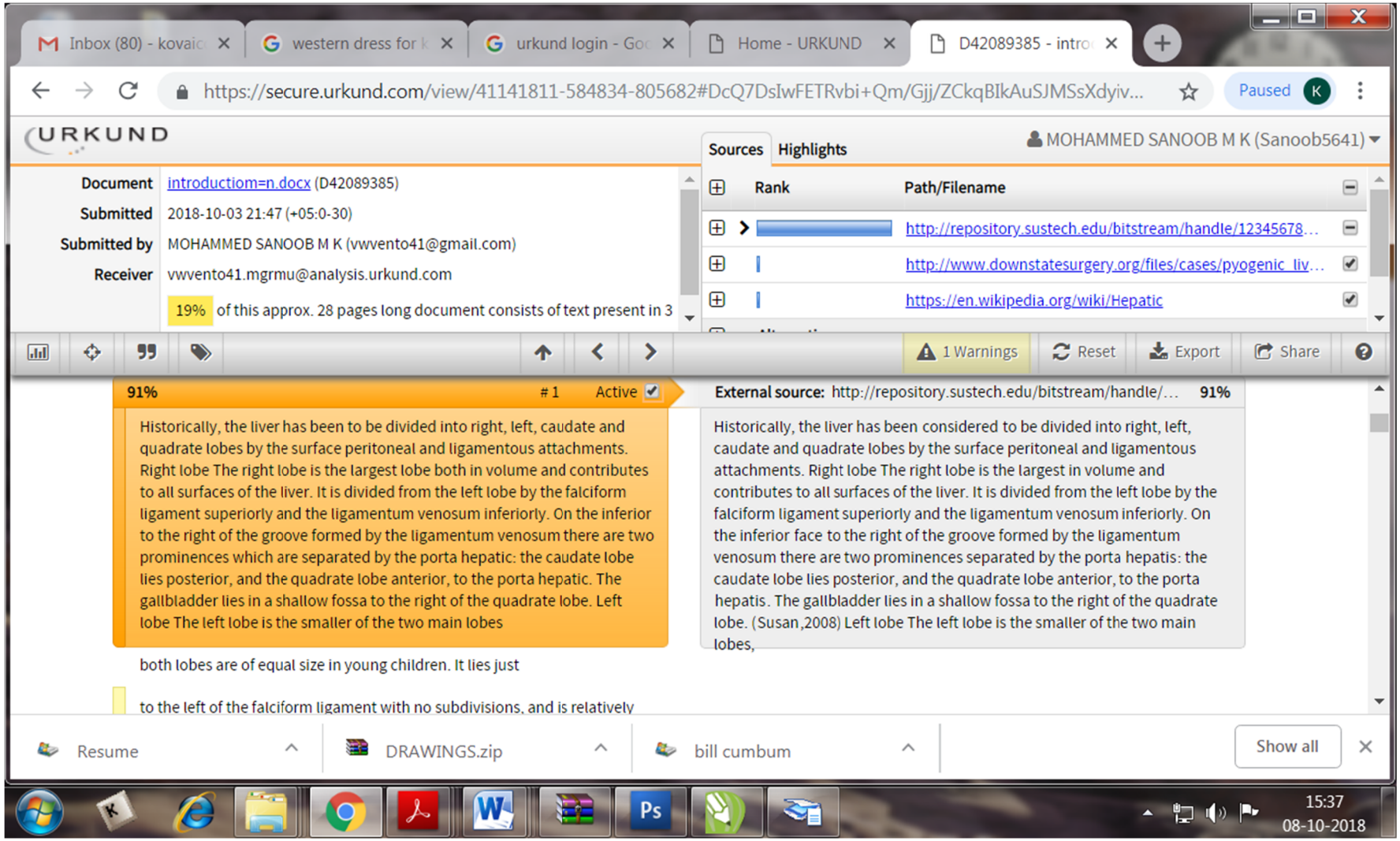
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Historically, the liver has been to be divided into right, left, caudate and quadrate lobes by the surface peritoneal and ligamentous attachments. Right lobe The right lobe is the largest lobe both in volume and contributes to all surfaces of the liver. It is divided from the left lobe by the falciform ligament superiorly and the ligamentum venosum inferiorly. On the inferior to the right of the groove formed by the ligamentum venosum there are two prominences which are separated by the porta hepatic: the caudate lobe lies posterior, and the quadrate lobe anterior, to the porta hepatic. The gallbladder lies in a shallow fossa to the right of the quadrate lobe. Left lobe The left lobe is the smaller of the two main lobes
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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled "**PROSPECTIVE ANALYSIS OF FACTORS DECIDING SURGICAL INTERVENTION IN LIVER ABSCESS** " is a genuine research work carried out by me under the guidance of Prof. Dr. S. Ganesh babu M.S, at Coimbatore medical college hospital, Coimbatore- 641018 during the period of my post graduate study for M.S, General Surgery from 2016 to 2019.

This dissertation is submitted to THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI in partial fulfilment of the requirements for the degree of M.S. General Surgery to be held in May 2019.

Dr. Mohammed Sanoob. M. K,

Post Graduate Student,

M. S. General Surgery,

Coimbatore Medical College Hospital.

ENDORSEMENT BY THE HOD AND HEAD OF THE INSTITUTION

This is to certify that the dissertation titled "**PROSPECTIVE ANALYSIS OF FACTORS DECIDING SURGICAL INTERVENTION IN LIVER ABSCESS"** is a bonafide research work done by **Dr. Mohammed Sanoob. M. K,** post graduate in M.S.General Surgery, Coimbatore Medical College, Coimbatore under the guidance of Prof. Dr. S. Ganesh babu M.S, during the period in 2014-2017 in partial fulfilment of the requirement for the award of M.S.General Surgery.

Prof. Dr.V.Elango,M.S,FAIS,
Professor and Head of the Department,
Dept. Of General Surgery,
Coimbatore Medical College Hospital.

Prof.Dr.B.Asokan ,M.S,Mch
Dean
Coimbatore Medical College Hospital.

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I express my thanks to my family members ,friends and all others who have helped me in the preparation of this dissertation.

Last but not least ; I heartily thank all the patients for their kind support without whom this study could ever have been done,

Dr. Mohammed Sanoob. M. K,

LIST OF ABBREVIATIONS

LFT	LIVER FUNCTION TESTS
USG	ULTRA SONOGRAM
TIPSS	TRANS INTRA HEPATIC PORTOSYSTEMATIC SHUNT
MODS	MULTIORGAN DYSFUNCTION SYNDROME ERCP ENDOSCOPIC RETROGRADE CHOLANGIO PANCREATOGRAM

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INTRODUCTION

Liver abscess has multimodal aetiological and risk factors, varied presentation, management approaches and high recurrence or residual disease.

Though it is common and elaborately studied, it is least understood and has pitfalls.

Hence planned to study the varying clinical and biochemical presentations of liver abscess, risk factors, management modalities, factors deciding necessity for surgical intervention and outcome.

Liver abscess is a significant problem of tropical countries and remains a great diagnostic and therapeutic problem. If left untreated, the disease invariably runs a lethal course. The management of this disease varies considerably from surgeon to surgeon. Liver abscess is a condition with significant mortality.

The most common presenting clinical symptoms are upper abdominal pain, high-grade fever, nausea, and vomiting. Loss of appetite, jaundice, and respiratory symptoms are less frequent clinical features. These clinical features are variable depending on the size of the abscess, general health of the patient, associated diseases, and complications.

The most common sign is right hypochondrial tenderness frequently with guarding and hepatomegaly. Some patients may present with jaundice, ascites, or pleural effusion. In majority of the cases, the underlying cause could not be identified.

Biliary tract disease is reported to be the most frequent cause followed by portal circulation, arterial circulation, cryptogenic, and trauma. It may be due to bacterial or parasitic invasion of liver. Majority of abscesses are multiple which are due to biliary system and arterial circulation and sub diaphragmatic and are noted in the right lobe of liver. Solitary or single abscess is due to portal circulation, cryptogenic, and trauma.

Early studies by Oschner et al. recommended open surgical drainage as the treatment of choice. For the past two decades, advances in the imaging field coupled with ultrasound-guided percutaneous needle aspiration and drainage brought dramatic changes in the pattern of treatment for pyogenic liver abscess.

The aim of our study was to determine the factors deciding necessity for surgical intervention and outcome.

As India is a one of the tropical country and home to 400 million people harbouring ,the causative organism of amoebic liver abscess ,it is important to thoroughly understanding of liver abscess

All these factor influenced me to select this topic which assumes more important in rural population .

Hence this study is designed to discuss the various factors deciding necessity of surgical intervention in liver abscess

OBJECTIVES OF STUDY

Aim:

1. To study the factors influencing surgical intervention in liver abscess.
2. To compare various treatment modalities in liver abscess.
3. To compare outcome in surgical and conservative treatment in liver abscess.

REVIEW OF LITERATURE

EMBRYOLOGY

Liver primodium appear on middle of third week as an out growth of endodermal epithelium at the distal end of foregut, just at its junction with the midgut. Liver, the largest gland in the body develops from three following sources

1. Parenchyma of liver from endodermal hepatic bud
2. Fibrous stroma derived from the mesenchyme of septum transversum
3. Sinusoids of liver develops from absorbed and broken vitelline and umbilical veins within the septum transversum

The outgrowth or liver bud has rapidly proliferating cells that penetrate the septum transversum (plate of intra embryonic mesoderm at the cranial edge of embryonic disc).

The liver bud soon divides into two parts, a large cranial part –pars hepatica and a small caudal part –pars cystica

Pars hepatica –forms the liver and Pars cystica forms the gall bladder and cystic ducts

The remaining bud just proximal to pars cystica forms the common bile duct

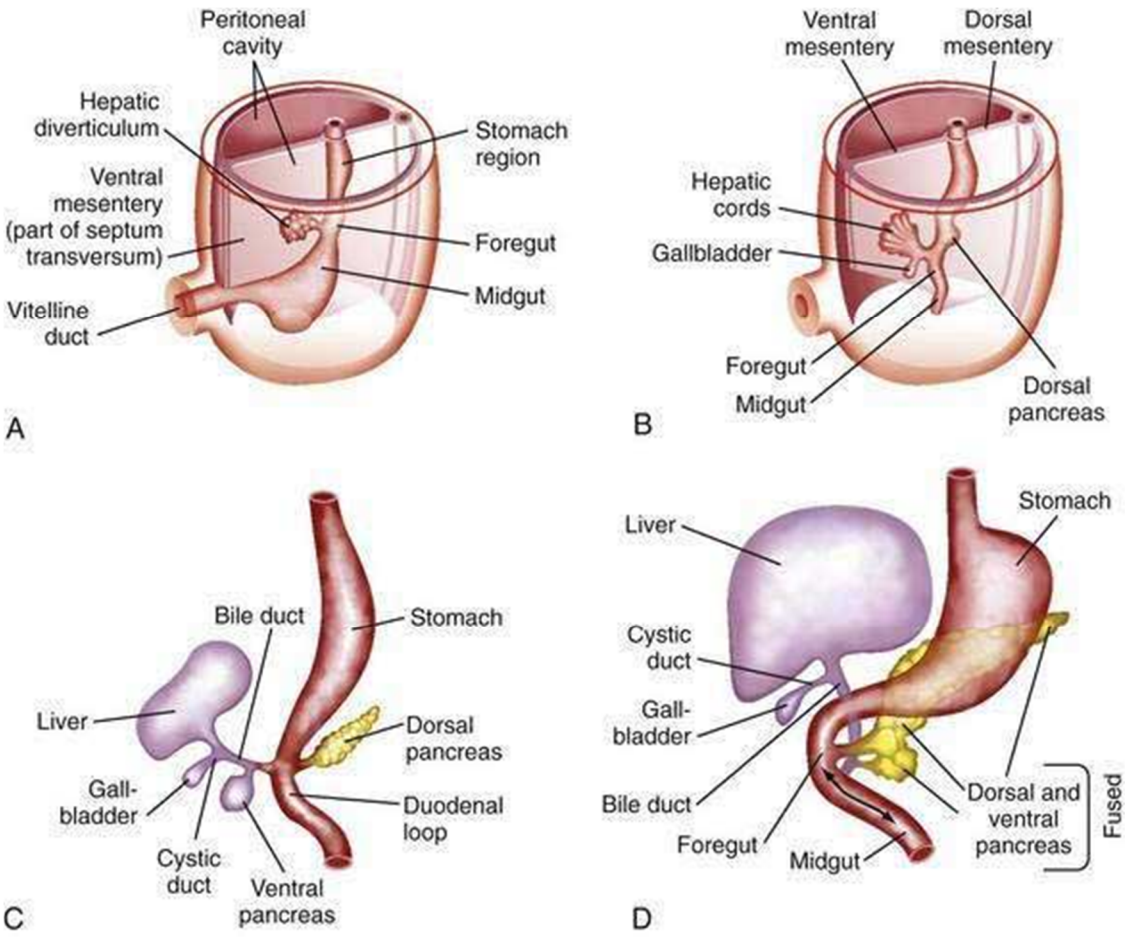
The fibrous stroma contains the hematopoietic cells, kupffer cells and connective tissue

The pars hepatica further divides into right and left lobes. Initially the right and left lobes are of equal size .As the right and left portions of pars

hepatica enlarge, they extend into the septum transversum .The cells arising from them form interlacing hepatic cords or cords of hepatocytes .The cells of hepatic cord arranged radially in hepatic lobules. The bile canaliculi and ductules are formed in liver parenchyma and later establish connections with extra hepatic bile ducts .Due to rapid enlargement of liver it occupies the major portions of abdominal cavity so gut herniates through umbilicus as physiological hernia, main reason for enlargement is oxygen rich blood supply and proliferation of hematopoietic tissue

The mesoderm on the surface of liver differentiates into visceral peritoneum excepts one cranial end, in this region the liver contact with rest of septum transversum.This ortion of septum is densely packed form the central tendon of diaphragm. The portion of liver in contact with diaphragm is not covered by peritoneum –bare area of liver

In 10^{th} week of development, the weight of liver is 10% of total body weight .Large nests of proliferating cells which produce red and white blood cells lie between hepatic cells anwalls of vessels .This activity gradually subsides during last 2 months of intra uterine life only small islands persist at birth, the weight of liver is only 5%of body weight



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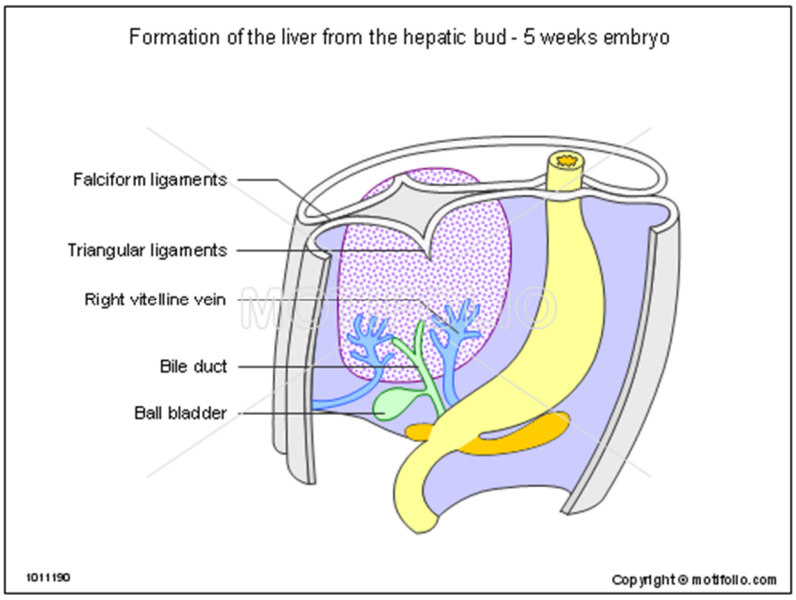

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ANATOMY

Historically, the liver has been to be divided into right, left, caudate and quadrate lobes by the surface peritoneal and ligamentous attachments.

Right lobe

The right lobe is the largest lobe both in volume and contributes to all surfaces of the liver. It is divided from the left lobe by the falciform ligament superiorly and the ligamentum venosum inferiorly. On the inferior to the right of the groove formed by the ligamentum venosum there are two prominences which are separated by the porta hepatic: the caudate lobe lies posterior, and the quadrate lobe anterior, to the porta hepatic. The gallbladder lies in a shallow fossa to the right of the quadrate lobe.

Left lobe

The left lobe is the smaller of the two main lobes both lobes are of equal size in young children.

It lies just to the left of the falciform ligament with no subdivisions, and is relatively thinner than the right lobe, having a thin apex that points into the left upper quadrant.

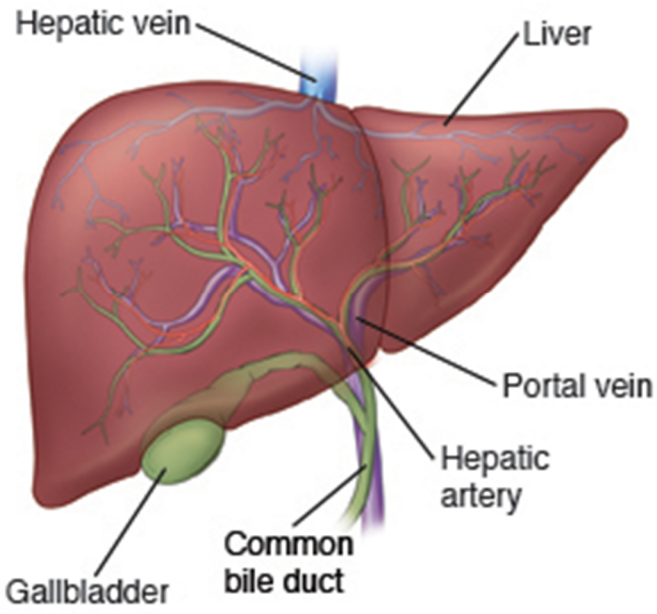
Quadrate lobe

The quadrate lobe is seen as a prominence on the inferior surface of the liver, to right of the groove which is formed by the ligamentum venosum (and it is functionally related to the left lobe although incorrectly related to right lobe). It lies just anterior to the porta hepatis and bounded by the gallbladder

fossa to the right, a short portion of the inferior border anteriorly, the fissure for the ligamentum teres to the left, and the porta hepatis posteriorly.

Caudate lobe

The caudate lobe is seen as a prominence on the inferior and posterior surfaces to the right of the groove is formed by the ligamentum venosum: it is posterior to the porta hepatis. To its right there is groove for the inferior vena cava. Above, it continues onto the superior surface on the right of the upper end of the fissure for ligamentum venosum. In gross anatomy it describes as this lobe is said to arise from the right lobe, but it is functionally separate



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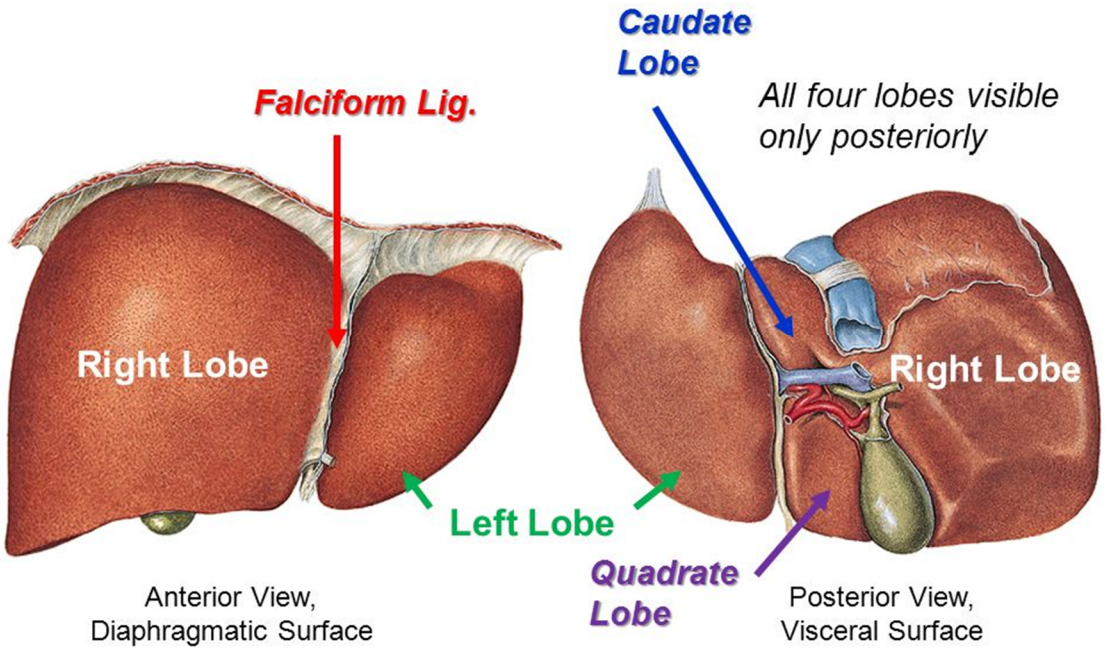

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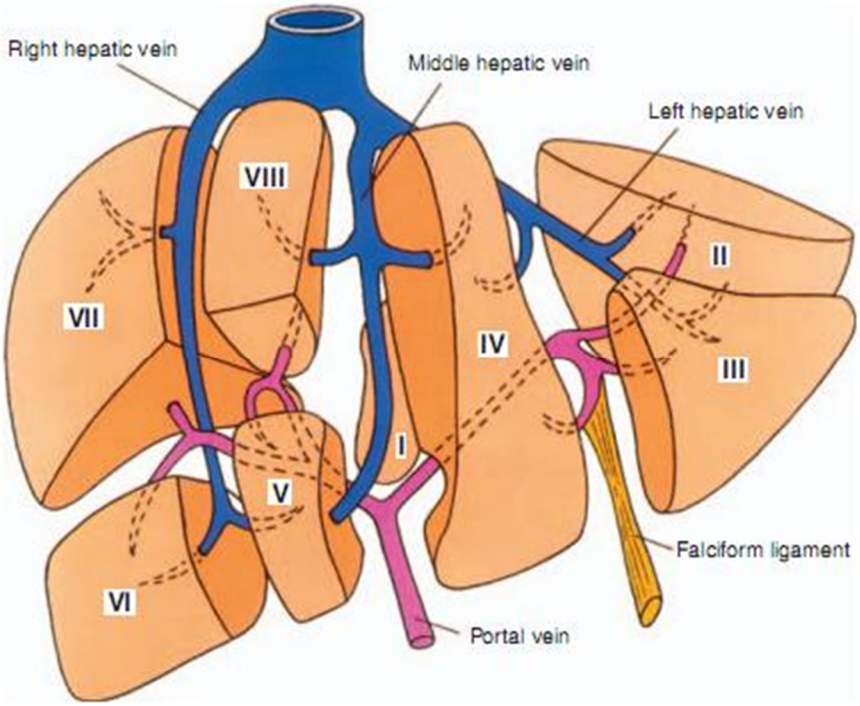
FUNCTIONAL ANATOMICAL DIVISIONS

The functional anatomy of the liver is based on Couinaud's division of the liver into eight (subsequently nine) functional segments, based upon the distribution of portal venous branches and the location of the hepatic veins in the parenchyma (Couinaud 1957). The understanding of the intrahepatic biliary anatomy, especially of the right ductal system, was enhanced by contributions from Hjortsjo (1948) and Healey & Schroy (1953) using the biliary system as the main guide for division of the liver



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The liver was divided into four portal sectors by four main branches of portal vein. These are right lateral, right medial, left medial and left lateral (sometimes the term posterior is used in place of lateral and anterior in place of medial). The three main hepatic veins which lie between these sectors are intersectorial veins. These intersectoral planes are named also as called portal fissures (scissures). The fissures which containing portal pedicles are called hepatic fissures. Each sector is sub-divided into segments (usually two) based on their supply by tertiary divisions of the vascular biliary sheaths.

Fissures of the liver

There are three major fissures, which is not visible on the surface, run through the liver parenchyma and contains the three main hepatic veins (main, left and right portal fissures). Three minor fissures are seen as physical clefts of the liver surface (umbilical, venous and fissure of Gans).

Main portal fissure

The main fissure which extends from the tip of gallbladder back to the midpoint of inferior vena cava and which contains the middle (main) hepatic vein. It divides the liver into right and left hemi-livers. Segments V and VIII lie to right and segment IV to the left of the fissure.

Left portal fissure

The left fissure which separates the left hemi-liver into medial (anterior) and lateral (posterior) sectors. Which extends from the midpoint of the anterior edge of the liver between the falciform ligament and the left triangular ligament to a point which marks as the confluence of the left and middle hepatic veins. As It contains the left hepatic vein and divides the left anterior and left posterior sectors: segment III just lies anteriorly and segment II posteriorly

Right portal fissure

The right portal fissure which divides the right hemi-liver into lateral (posterior) and medial (anterior) sectors. The plane of right fissure is usually the most variable amongst the main fissures and it runs approximately diagonally through the gross right lobe from lateral end of anterior border to confluence of the left side and the middle hepatic veins. The fissure which divides the right anterior sector to its left (segments V and VIII) from the right posterior sector to its right (segments VI and VII), and which usually contains the right hepatic vein. The right fissure which marks as the thickest point of liver parenchyma which is commonly transected while liver resection.

Umbilical fissure

Umbilical fissure is the one which separates segment III from segment VI within the left anterior sector and which contains a main branch of the left hepatic vein (the umbilical fissure vein). It is pointed out by the attachment of the falciform ligament and sometimes it gets covered by the ridge of liver tissue which is extending between the segments: it is very often avascular and which can be divided safely with a diathermy during the surgical approach. It has the umbilical portion of the left portal vein and the final divisions of the left hepatic duct and the left hepatic artery branches. Umbilical portion of the left portal vein is an important landmark: access to this vein and during mobilization of the left portal vein is one of the essential steps in surgery for hilar cholangiocarcinoma. To know the arrangement of the portal vein, hepatic artery and bile duct within the umbilical fissure is also needed when splitting the liver for an adult and paediatric recipient and for live donor liver transplantation for a child recipient.

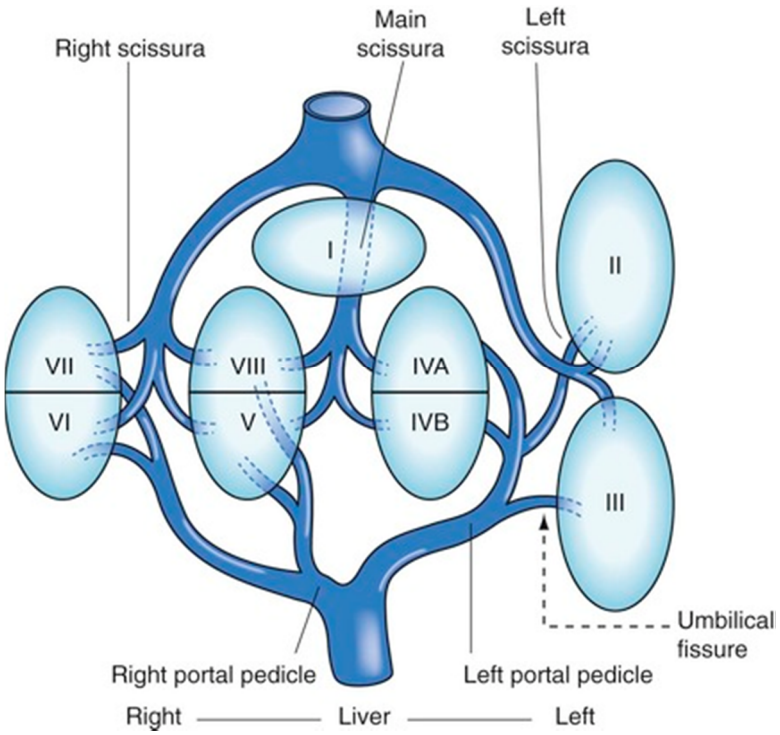
Venous fissure

The venous fissure is direct continuation of the umbilical fissure on the under surface of the liver and usually it contains the ligamentum venosum. It lies between the caudate lobe and segment IV. Deeper continuation of this plane is the dorsal fissure.

Fissure of Gans

Fissure of Gans is another fissure which lies on the undersurface of the right lobe of the liver behind the gallbladder fossa. It usually contains the portal

pedicle to the right posterior sector and is thought to correspond to the right fissure as it relates to the separation of the sectors of the liver.



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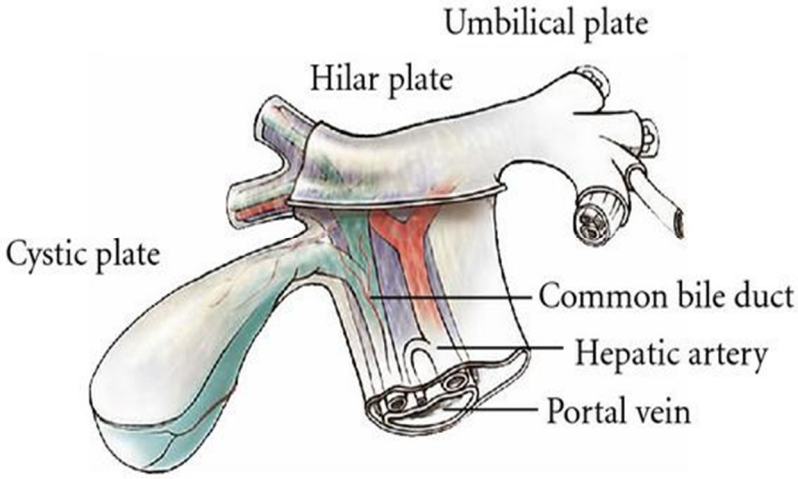

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Sectors and segments of the liver

Sectors

The sectors of the liver are made up of between one and three segments: right lateral sector = segments VI and VII; right medial sector = segments V and VIII; left medial sector = segments III and IV (and part of I); left lateral sector = segment II. Segments are numbered in an ante-clockwise spiral centered on the portal vein with the liver viewed from below, starting with segment I up to segment VI, and then back clockwise for the most cranial two segments VII and VIII.

Segment I

Segment I represents the anatomical caudate lobe and lies posterior (dorsal) to segment IV with its left half directly posterior to segments II and II and its medial half surrounded by major vascular branches. Glissonian sheaths for segment I arise from both right and left main sheaths: the segment therefore receives the vessels independently from the left and right portal veins and hepatic arteries, and usually drains independently into the inferior vena cava through multiple small branches (referred to as the lower group). They usually arise in the lower and sometimes from the middle third, but do not ever from the upper third of the segment. The bile ducts draining these segments are closely related to the confluence of the right and left hepatic ducts such that excision of central bile duct tumours usually requires removal of segment IV.

Segment II

Segment II is the only segment of the left lateral sector of the liver and which lies postero-lateral to the left fissure. It usually has only one Glissonian sheath and drains into the left hepatic vein.very Rarely, a separate vein drains directly into the inferior vena cava.

Segment III

Segment III which lies between the umbilical fissure and left fissure and is mostly supplied by one to three Glissonian sheaths: majorly drains into the left hepatic vein. The vein of the falciform ligament can provide an alternative drainage route for segment III.

Segment IV

Segment IV which lies between the umbilical fissure and the main fissure, anterior to the dorsal fissure and segment I. Segment IV has supply of three to five Glissonian sheaths, of which the majority arise mostly in the umbilical fissure; their origins are close to those that supply to segments II and III. Some times segment IV is supplied by branches from the main left pedicle. The main venous drainage segment into the middle hepatic vein; the segment can also drain into the left hepatic vein through the vein of the falciform ligament.

Segment V

Segment V is the inferior segment of the right medial sector and occupies between the middle and the right hepatic veins. Size is variable, as are the number of Glissonian sheaths that supply it also variable . Venous drainage mostly into the right and middle hepatic veins.

Segment VI

Segment VI which forms the inferior part of the right lateral sector posterior to the right portal fissure. Mostly supplied by two to three branches from the right posterior Glisson's sheath, but sometimes the Glisson's sheath to segment VI can arise directly from the right pedicle. Venous drainage into the right hepatic vein, also sometimes via the right inferior hepatic vein directly into the inferior vena cava.

Segment VII

Segment VII forms the superior part of the posterior sector and occupies behind the right hepatic vein. The sheaths to this segment VII are mostly single. The venous drainage into the right hepatic vein; sometimes the segment can drain through the right middle hepatic vein directly into the inferior vena cava.

Segment VIII

Segment VIII forms the superior part of the right anterior sector. The right anterior sectoral sheaths end in segment VIII and supply it after giving off branches to segment V. The venous drainage into the right and middle hepatic veins.

Segment IX

Segment IX is a newly added subdivision of segment I, and describes that part of the segment that lies posterior to segment VIII.

Supports of the liver

The liver is stabilized and its position maintained by the right upper quadrant of the abdomen by both static and dynamic factors. A three-tier classification of the anatomical factors: the suspensory attachments at the posterior abdominal wall to the inferior vena cava, hepatic veins, coronary and triangular ligaments (primary factors); the support given by the right kidney, right colonic angle and duodenopancreatic complex (secondary factors); the attachment to the anterior abdominal wall and diaphragm by the falciform ligament (tertiary factors)

The inferior vena cava and the supra-hepatic veins, mostly the right hepatic vein, found to be the most important anatomical structures that support the bulk of the liver. Other factors which is responsible for the maintenance of the position of the liver within the abdominal cavity include positive intra-abdominal pressure and the movement of the diaphragm during respiration.

Peritoneal attachments and ligaments of the liver

The liver is attached to the anterior abdominal wall, diaphragm and other viscera by several ligaments formed from condensations of the peritoneum

Falciform ligament

Liver attached in front to the anterior abdominal wall by the falciform ligament. The two layers of falciform ligament descend from the posterior surface of the anterior abdominal wall and diaphragm and turn onto the anterior and superior surfaces of the liver. On the dome of the superior surface, the right

leaf of ligament runs laterally and is continuous with the upper layer of the coronary ligament. The left layer of the falciform ligament takes a medial turn and is continuous with the anterior layer of the left triangular ligament. The ligamentum teres, which represents as obliterated left umbilical vein, runs over lower free border of the falciform ligament and continues into a fissure on the inferior surface of the liver. In fetal life the left umbilical vein opens into the left portal vein: and is supposed to be obliterated in adult life, but frequently remains partially patent. This lumen sometimes open up in conditions such as portal hypertension to form a collateral channel. The ligamentum teres is important in abdominal surgery for several reasons. It is quite often divided in upper abdominal surgery to optimize access to the upper abdominal viscera or required as the first step in the mobilization of the liver. The ligament mostly vascularized by numerous arterial branches, majority from the segment IV artery, and these form an anastomotic connection with the branches of the internal thoracic artery: and therefore important to ligate or coagulate the ligament during its division. The ligamentum teres used as a landmark and guide to the segment III hepatic duct used in hepatocojejunostomy formation, and to the left portal vein lying in the umbilical fissure during creation of a mesentericoportal shunt.

Coronary ligament

The coronary ligament mostly formed by the reflection of the peritoneum from the diaphragm onto the posterior surfaces of the right lobe of the liver.there is a large triangular area of liver devoid of peritoneal covering,

the ‘bare area' of the liver, is defined between the two layers of the coronary ligament. Here the liver is attached to the diaphragm by areolar tissue and is in continuous inferiorly with the anterior pararenal space. The coronary ligament is continunity on the right with the right triangular ligament. On the left, the two layers become closely related , and form the left triangular ligament. The topmost layer of the coronary ligament is reflected superiorly onto the inferior surface of the diaphragm and inferiorly onto the right and superior surface of the liver. The lowermost layer of the coronary ligament is reflected inferiorly over the right suprarenal gland and right kidney, and superiorly onto the inferior surface of the liver. Surgical division of the right triangular and coronary ligaments help us the right lobe of the liver to be brought forward, and exposes the lateral aspect of the inferior vena cava behind the liver

Triangular ligaments

The left triangular ligament -double layer of peritoneum which extends to a variable length over the superior border of left lobe of the liver. Medially the anterior leaf is in continuity with the left layer of the falciform ligament and the posterior layer is continuous with the left layer of the lesser omentum. The left triangular ligament occupiesin the front of the abdominal part of the oesophagus, the uppermost end of the lesser omentum and part of the fundus of the stomach. Division of this left triangular ligament allows the left lobe of the liver to be mobilized for exposure of the abdominal oesophagus and crura of the diaphragm. The left triangular ligament act as a important stabilizing factor for the left lobe in operations involving removal of the right lobe of the

liver. Normally Its division will result in the left lobe being unstable to the extent that it can rotate and displace into the space vacated under the right hemidiaphragm: and this much extreme degree of rotation can compromise the venous outflow of the liver with consequent liver dysfunction. unknowly If it is divided, the left triangular ligament must be fixed at the end of the surgery, in addition to fixing back the falciform ligament.

The right triangular ligament is a short structure which occupies apex of the ‘bare area' of the liver and is continuous with the layers of the coronary ligament

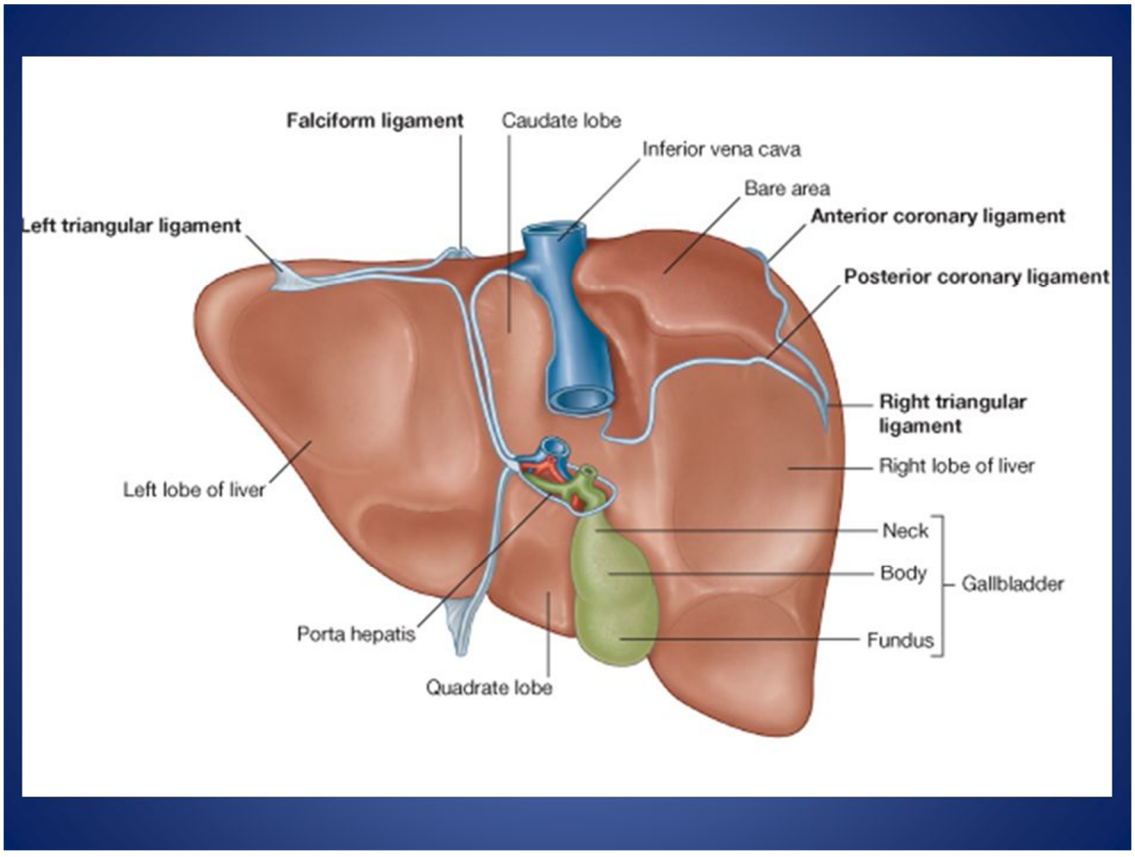

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Figure : 8

Lesser omentum

The lesser omentum derived from fold of peritoneum which extends from the lesser curve of stomach and proximal duodenum to the inferior surface of the liver. And its attachment to the inferior surface of the liver is L-shaped. The vertical component follows the line of the fissure for the ligamentum venosum, the fibrous remnant of the ductus venosus. Most inferiorly the attachment runs horizontally to complete the L in the porta hepatis. At its uppermost end, the superior layer of the lesser omentum is continuous on the left with the posterior layer of the left triangular ligament, and inferior layer is continuous on the right with the coronary ligament as it encloses the inferior vena cava. At its lowermost end, the two layers diverge to surround the structures of the porta hepatis. Avery thin fibrous condensation of fascia usually runs from the medial end of the porta hepatis into the fissure in the inferior surface which contains the ligamentum teres. This fascia which is in continuous with the lower border of the falciform ligament when the ligamentum teres re-emerges at the inferior border of the liver. Uttermost Care should be taken when dividing the lesser omentum, because chance of an aberrant left hepatic artery may run in the medial end should be in mind: when present, it enormously extends to the liver at the base of the umbilical fissure, and may be identified by a pulsation in the lesser omentum close to the umbilical fissure

Ligamentum venosum

The ligamentum venosum is the obliterated venous connection that existed between the left portal vein and the left hepatic vein in fetal life. It acts

as a guide and aid to control the left hepatic vein extrahepatically during surgeryof liver. By dividing this ligament close to its insertion into the left hepatic vein and retracting it laterally, the angle which lies between the left and the middle hepatic veins, required for dissection and control of the left hepatic vein, may be accessed.

Porta hepatis, hepatoduodenal ligament and hilar plate

The porta hepatis represent as deep fissure on the inferior surface of the liver. It lies between the quadrate lobe in front and the caudate process behind, and contains the portal vein, hepatic artery and hepatic nervous plexuses as it ascend into the parenchyma of the liver, and the right and left bile ducts and some lymph vessels as they rise from the liver. The hepatic ducts which lie anterior to the portal vein and its branches, and the hepatic artery with its branches lies between the two. All these structures are covered by the perivascular fibrous capsule, the hepatobiliary capsule of Glisson, a sheath of loose connective tissue which surrounds the vessels as they course through the portal canals in the liver and is also continuous with the fibrous hepatic capsule. The dense aggregation of vessels, supporting connective tissue, and liver parenchyma just above the porta hepatis is called as the ‘hilar plate' of the liver. For Understanding of the concept of hilar plate is important in surgical approaches to the hilar structures. Division or lowering of the hilar plate are important for surgical access to the left hepatic duct. The hepatic artery, bile duct and portal vein which extend from the porta hepatis towards the duodenum around the free edge of the hepatoduodenal ligament, which forms

the anterior boundary of the epiploic foramen. For Rapid control of the vessels entering the porta hepatis can be obtained by dividing the lesser omentum to the left of these structures and passing a tape around them from left to right through the epiploic foramen (a ‘Pringle' manoeuvre).

The left hepatic duct which remains extrahepatic as it runs down to the bifurcation along the base of segment IV (the quadrate lobe). This extrahepatic length of duct is mostly useful when performing high biliary duct reconstructions where a length of jejunum is anastomosed to form a biliary-enteric bypass, to treat strictures of the common hepatic duct.

Glisson's sheath

Glisson's capsule form as condensations around the branches of the portal triad structures as Glissonian sheaths as they enter the liver parenchyma and extends as far the individual segments of the liver. This arrangement helps in surgical control of both the main right and left pedicles of the liver as well as all the structures (biliary, autonomic and lymphatic elements) that supply individual sectors and segments in liver complex resectional surgery.

SURFACES OF THE LIVER

The liver is divided superior, anterior, right, posterior and inferior surfaces, and has a distinct inferior border. However, the superior, anterior and right surfaces are continuous and no definable borders separate them. It will better to group them as the diaphragmatic surface, which is mostly separated from the inferior or visceral surface, by a narrow inferior border. At the infrasternal angle the inferior border is relation to the anterior abdominal wall

and is accessible to examination by percussion, but is not usually palpable. In the midline, the inferior border of the liver is close to the trans pyloric plane, about a hand's breadth below the xiphisternal joint. In women and children the border often projects a little below the right costal margin.

Superior surface

The superior surface is largest surface and occupies immediately below the diaphragm, separated from it by peritoneum except for a minute triangular area where the two layers of the falciform ligament diverge. The majority of the superior surface lies beneath the right dome, but there is a shallow cardiac impression centrally that corresponds to the position of the heart above the central tendon of the diaphragm. The left side of the superior surface lies beneath part of the left dome of the diaphragm.

Anterior surface

The anterior surface mostly triangular and convex and is covered by peritoneum except at its attachment of the falciform ligament. Much of it is in contact with the anterior attachment of the diaphragm. On the right the diaphragm which separates it from the pleura and sixth to tenth ribs and cartilages, and on the left from the seventh and eighth costal cartilages.

Right surface

The right surface is covered by a layer peritoneum and lies adjacent to the right dome of the diaphragm that separates it from the right lung and pleura and the seventh to 11th ribs. The right lung and basal part of pleura both lie above and lateral to its upper third, between the diaphragm and the seventh and

eighth ribs. The diaphragm, which has costodiaphragmatic recess lined by pleura, and the ninth and tenth ribs all lie lateral to the middle third of the right surface. Lateral to the lower third, the diaphragm and thoracic wall are in direct contact with liver . Rarely, the hepatic flexure and proximal transverse colon may lie on a long mesentery over the right and superior surfaces of the liver, referred to as Chilaiditi syndrome.

Posterior surface

The posterior surface is mostly convex, wide on the right, but narrow on the left. There is a deep median concavity corresponds to the forward convexity of the vertebral column close to the attachment of the ligamentum venosum. Large part of the posterior surface is attached to the diaphragm by loose connective tissue, forming the triangular ‘bare area'. The inferior vena cava which lies in a groove or tunnel in the medial end of the ‘bare area'. To the left of the inferior venecaval groove the posterior surface of the liver is formed by the caudate lobe, and covered by a layer of peritoneum continuous with that of the inferior layer of the coronary ligament and the layers of the lesser omentum. The caudate lobe is in relation to the diaphragmatic crura above the aortic opening and the right inferior phrenic artery, and separated by these structures from the descending thoracic aorta.

The fissure for the ligamentum venosum which separates the posterior aspect of the caudate lobe from the main part of the left lobe. The fissure that cuts deeply in front of the caudate lobe and contains the two layers of the lesser omentum. The posterior surface over the left lobe had a shallow impression

near the upper end of the fissure for the ligamentum venosum that is caused by the abdominal part of the oesophagus. The posterior surface of the left lobe to the left of this impression is in relation to part of fundus of stomach together with posterior relation referred as bed of liver

Inferior surface

The inferior surface is bounded by the inferior edge of the liver. It combines with the posterior surface in the region of the origin of the lesser omentum, the porta hepatis and the lower layer of the coronary ligament, and is marked near the midline by a sharp fissure which contains the ligamentum teres (the obliterated fetal left umbilical vein). The gallbladder which occupies the shallow fossa, but this is variable: it may have a short mesentery or be completely intrahepatic and lie within a cleft in the liver parenchyma. The quadrate lobe lies between the fissure for the ligamentum teres and the gallbladder.

The inferior surface of the left lobe is inferiorly related to the fundus of the stomach and the upper lesser omentum. The quadrate lobe which lies adjacent to the pylorus, first part of the duodenum and the lower part of the lesser omentum. Sometimes the transverse colon lies between the duodenum and the quadrate lobe. To the right of the gallbladder, the inferior surface is in relation with the hepatic flexure of the colon, the right suprarenal gland and right kidney, and the first part of the duodenum.

VASCULAR SUPPLY AND LYMPHATIC DRAINAGE

The vessels in relation with the liver are the portal vein, hepatic artery and hepatic veins. The portal vein and hepatic artery ascend runs in the lesser omentum to the porta hepatis, where each of them bifurcates. The hepatic bile duct and lymphatic vessels descend from the porta hepatis in the same omentum . The hepatic veins leave the liver through its posterior surface and drains into the inferior vena cava.

Hepatic artery

In adults the hepatic artery is of intermediate size between the left gastric and splenic arteries sizes. In fetal and early postnatal life it is one of the largest branches of the coeliac axis. The hepatic artery gives off right gastric, gastroduodenal and cystic branches as well as direct branches to the bile duct from the right hepatic and sometimes the supraduodenal artery .just After its origin from the coeliac axis, the hepatic artery passes anteriorly and laterally below the epiploic foramen to the upper aspect of the first part of the duodenum. It may be subdivided into the common hepatic artery, from the coeliac trunk to the origin of the gastro duodenal artery, and the hepatic artery ‘proper', from that point to its bifurcation. It passes just anterior to the portal vein and ascends anterior to the epiploic foramen between the layers of the lesser omentum. Within the free edge of the lesser omentum the hepatic artery and lies medial to the common bile duct and anterior to the portal vein. At the porta hepatis it usually divides into right and left branches before these run into the parenchyma of the liver. The right hepatic artery usually crosses posterior

(occasionally anterior) to the common hepatic duct . This close proximity relates us that the right hepatic artery is involved in bile duct cancer earlier than the left hepatic artery. Sometimes the right hepatic artery crosses in front of the common bile duct and may be injured in surgery of the common bile duct. It mostly always divides into an anterior branch supplying segments V and VIII, and a posterior branch supplying segments VI and VII. The anterior division mostly supplies a branch to segment I and the gallbladder. The segmental arteries are macroscopically end-arteries although some collateral circulation occurs between segments via fine terminal branches.

A little number of normal variants are important to demonstrate angiographically because they may influence our surgical and interventional radiological procedures. A vessel which supplies a lobe in addition to its normal vessel is defined as an accessory artery. A replaced hepatic artery means a vessel that does not originate from an orthodox position and provides the sole supply to that lobe. Rarely a replaced common hepatic artery arises from the superior mesenteric artery and is identified at surgery by a relatively superficial portal vein (reflecting the absence of a common hepatic artery that would normally cross in front of the vein). Most commonly a replaced right hepatic artery or an accessory right hepatic artery arises from the superior mesenteric artery . In this scenario they run behind the portal vein and bile duct in the lesser omentum and can be identified at surgery by pulsation behind the portal vein. The accessory right hepatic artery might be injured during resections of the pancreatic head because the artery lies in close proximity to

the portal vein. Occasionally, a replaced left hepatic artery or an accessory branch arises from the left gastric artery: these vessels provides a source of collateral arterial circulation in cases of occlusion of the vessels in the porta hepatis but may also be injured during mobilization of the stomach as it lies in the upper portion of the lesser omentum. sometimes, accessory left or right hepatic arteries may arise from the gastroduodenal artery or aorta. The presence of replaced arteries can be lifesaving in patients with bile duct cancer: because they are further away from the bile duct they tend to be spared from the cancer, making excision of the tumour feasible.

Some Variations in the intrahepatic arteries are common and may be surgically important. The example, the segment VI artery most commonly arises from the left hepatic artery, approximately 10–20% of cases it arises from the right hepatic artery or the main hepatic artery.if we fail to recognize this variation may compromise the blood supply to segment IV following right hepatectomy, and is especially important following right lobe donation for live donor liver transplantation.

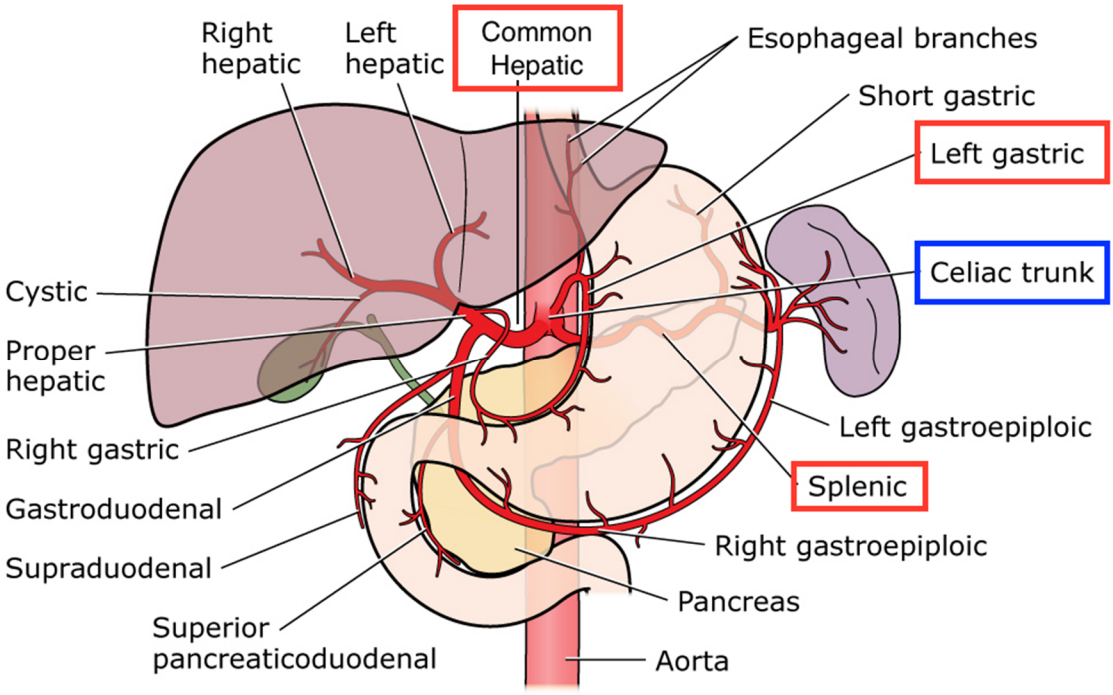


Figure : 9

Veins

The liver has two venous systems. The portal system conveys venous blood from the majority of the gastrointestinal tract and its associated organs to the liver. The hepatic venous system drains blood from the liver parenchyma into the inferior vena cava.

Portal vein

The portal vein starts at the level of the second lumbar vertebra and is formed from the convergence of the superior mesenteric and splenic vein. It is about 8 cm long and lies anterior to the inferior vena cava and posterior to the neck of the pancreas. It lies obliquely to the right and ascends behind the first part of the duodenum, the common bile duct and gastroduodenal artery.Mostly, At this point it is directly anterior to the inferior vena cava. It enters the right border of the lesser omentum, ascends anterior to the epiploic foramen to reach

the right end of the porta hepatis and then divides into right and left main branches which accompany the corresponding branches of the hepatic artery into the liver. In the lesser omentum the portal vein usually lies posterior to both the common bile duct and hepatic artery. It is surrounded by the hepatic nerve plexus and accompanied by many lymph vessels and some lymph node

The main extrahepatic tributaries of the portal vein are the coronary or the left gastric vein, which usually ends in the left margin of the portal vein, and the posterior superior pancreatoduodenal vein nearer to the head of the pancreas. The portal vein mostly divides into right and left branches at the hilum . The left portal vein has a longer extraparenchymal course fo about 4–5 cm and tends to lie slightly more horizontally than the right portal vein, but is often of smaller calibre. It has horizontal and vertical portions.

The horizontal portion which runs along the base of segment VI and often gives branches to segment I and sometimes to segment VI in this part of its course. The branch to segment II continues laterally but the main left portal vein takes a more anterior and vertical course in the umbilical fissure (the vein of the umbilical fissure) where it gives branches to segments III and IV and receives the obliterated left umbilical vein (ligamentum teres).

The majority of the supply for segment IV comes from the left portal vein, and only sometimes from the right via proximal branches of the main vein or branches from veins to segments V or VIII. The right portal vein is about 2–3 cm in length and usually divides into a right medial (anterior) sectoral division supplying segments V and VIII, and a right lateral (posterior)

sectoral division supplying segments VI and Variations mostly involve the right portal vein: absence of a right portal vein with the resulting portal trifurcation in the form of left portal vein, right medial and right lateral portal veins, is present in 10–15% of livers.

Occasionally the right medial vein which arises from the left portal vein, a variant which is important to remember during left sided liver resection. The portal trifurcation have implications for split liver and live donor liver transplantation, where its presence might be considered as a relative contra-indication. In some rare occasions, the portal bifurcation is absent, in which case the main portal vein enters the liver giving off the right segmental branches and then turns left to supply the left lobe of the liver (a contraindication to major liver surgery). Occasionally one or more of the segmental branches of the right lobe (especially segment IV) arises proximally.

Porto-systemic shunts

Due to Increased pressure within the portal venous system may result in dilatation of the portal venous tributaries: a reversal of flow may occur where these veins form anastomoses with veins which drain into the systemic venous circulation. The common sites where we usually come across porto-systemic shunts may occur, and the associated clinical implications

Table : 1

Portal veins	Systemic veins	Clinical presentations
Left gastric and lower oesophageal veins	Lower branches of oesophageal veins that drain into azygos and accessory hemiazygos veins	Oesophageal or gastric varices
Superior rectal veins	Middle and inferior rectal veins that drain into internal iliac and pudendal veins	Rectal varices
Persistent tributaries of left branch of portal vein, running in ligamentum teres	Peri-umbilical branches of superior and inferior epigastric veins	'Caput medusae'
Intraparenchymal branches of right branch of portal vein, lying in liver tissue exposed in 'bare area'	Retroperitoneal veins that drain into lumbar, azygos and hemiazygos veins	Retroperitoneal dilated veins are at risk during surgery or interventional procedures
Omental and colonic veins in the region of the hepatic and splenic flexure	Retroperitoneal veins in the region of the hepatic and splenic flexure	
Patent ductus venosus connected to the left branch of the portal vein	Inferior vena cava	Is rare

Hepatic veins

The liver is drained mainly by three major hepatic veins into the suprahepatic part of the inferior vena cava and a multitude of minor hepatic

veins that drain into the intrahepatic inferior vena cava. The three major veins are located between the four major sectors of liver

Right hepatic vein

This one is the longest and the largest vein and also the most variable. It is mostly single, but sometimes it remains as two trunks until it terminates by draining into the inferior vena cava. The right hepatic vein runs into the intersectoral plane between the right medial and right lateral sectors. It drains the whole of segments VI and VII and parts of segments V and VIII. The extent of it's the contribution to the drainage of segments V and VIII is variable, and depends upon the size of the veins that drain these segments into the middle hepatic vein. The right hepatic vein is formed near the anterior and inferior edge of the liver and which lies in a coronal plane through most of its course. It drains into the inferior vena cava near the upper border of the caudate lobe. While considering Of all the three major veins the right hepatic vein is the one which is the most variable in its size not only due to the differential contribution to segments V and VIII drainage along with the middle hepatic vein but also due to the occasional presence of an accessory right middle and right inferior veins.

Middle hepatic vein

The middle hepatic vein which lies along the plane between the right medial and the left medial sectors of the liver. It usually joins with the left hepatic vein and terminates into the inferior vena cava as a common trunk. It drains mostly the central part of the liver receiving constant branches from

segments IV, V and VIII. The sizes of the branches draining segments V and VIII are variable and are of surgical importance in terms of right lobe living donation.

Left hepatic vein

The left hepatic vein usually lies between the left medial and left lateral sectors of the liver. It drains segments II, III and occasionally IV. Small veins draining segment II and occasionally the superior part of segment IV may drain directly into the inferior vena cava in the minority of livers. Some times a major tributary of the left hepatic vein, the umbilical fissure vein runs in the intersegmental plane between segments III and IV and contributes to their drainage. Occasionally the vein draining segment III ends separately in the confluence of the left and middle hepatic veins. Those variations in the venous drainage are of significance in terms of split liver transplantation and live donor liver transplantation.

Minor veins

Segment I veins usually drain directly into the inferior vena cava and vary in number from one to five. it has an independent drainage from the rest of the liver in patients with Budd–Chiari syndrome, where all the major hepatic veins are blocked, segment I often continues to drain effectively and undergoes compensatory hypertrophy.very Rarely there is an accessory right middle or inferior hepatic vein. When they are present they are of surgical importance especially if they are of more than 5 mm in diameter; they drain segments V and VI independently of the three major veins and therefore any tumour

involving the three major veins can be safely resected as long as venous drainage from the accessory veins is ensured. In live donor and split liver transplantation these veins must be individually anastomosed to the recipient inferior vena cava to ensure adequate venous drainage.

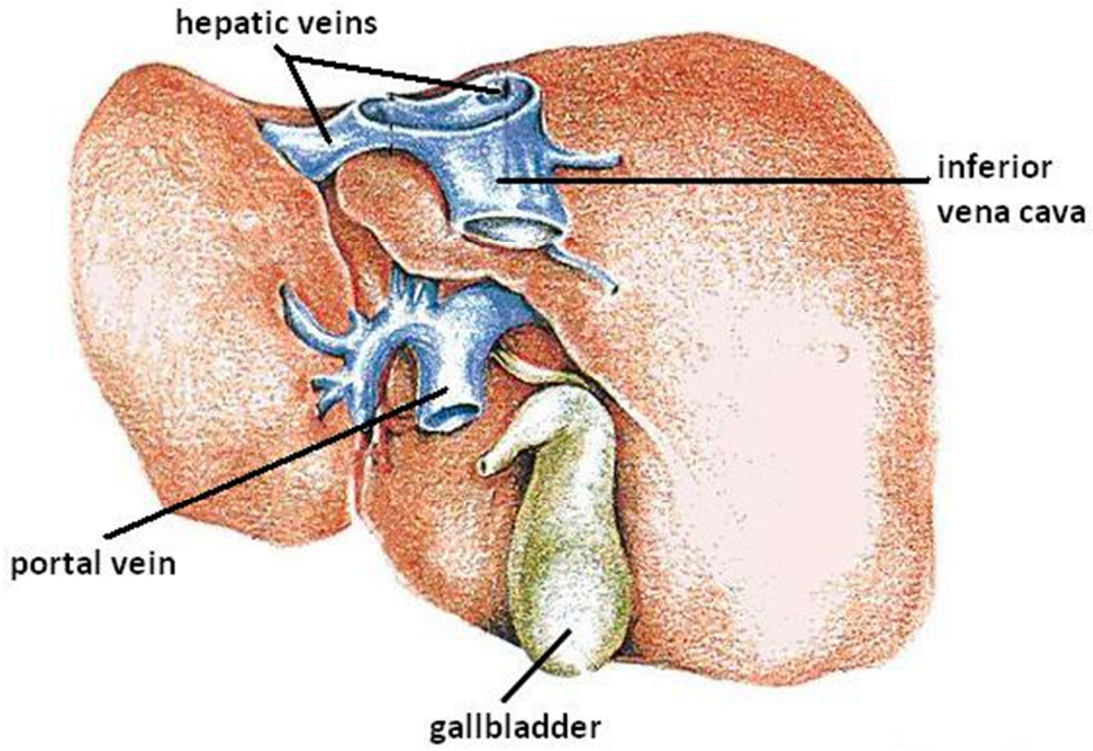


Figure : 10

Transjugular intraparenchymal porto-systemic shunt (TIPS) procedure for portal hypertension

In extreme cases of chronic portal hypertension, large calibre anastomoses between portal and systemic circulations may be formed within the liver parenchyma using a balloon catheter, introduced via the internal jugular vein and under radiological control, to puncture across and rupture through a thinned strip of liver parenchyma between hepatic veins and dilated portal branches.

Lymphatics

Lymph from the liver has abundant protein content. Lymphatic drainage of the liver is wide and may pass to nodes above and below the diaphragm. When there is Obstruction of the hepatic venous drainage increases the flow of lymph in the thoracic duct. Hepatic collecting vessels are divided into superficial and deep systems

Superficial hepatic vessels

The superficial vessels run mostly in the subserosal areolar tissue over the whole surface of the liver and drain in four directions. Lymph vessels which drain from the majority of the posterior surface, the surface of the caudate lobe, and the posterior part of the inferior surface of the right lobe, accompany the inferior vena cava and drain into pericaval nodes. Most of the Vessels in the coronary and right triangular ligaments may directly enter the thoracic duct without any intervening node. Vessels from the majority of the inferior surface, anterior surface and most of the superior surface all of which converge on the porta hepatis to drain into the hepatic nodes. A few lymph vessels from the posterior surface of the lateral end of the left lobe pass towards the oesophageal opening to drain into the para-cardiac nodes.usually One or two lymph trunks from the right surface and right end of the superior surface accompany the inferior phrenic artery across the right crus to drain into the coeliac nodes.

Deep hepatic vessels

The most of the liver parenchyma is drained by lymphatic vessels within the substance of the liver. Fine lymphatic vessels merge to form larger vessels. Some of which run superiorly through the parenchyma to form the ascending trunks which accompany the hepatic veins and pass through the caval opening in the diaphragm to drain into nodes around the end of the inferior vena cava. Vessels from the lower portion of the liver form descending trunks which emerge from the porta hepatis and drain into the hepatic nodes.

INNERVATION

The liver usually have a dual innervation. The parenchyma is supplied by hepatic nerves which arise from the hepatic plexus and contain sympathetic and parasympathetic (vagal) fibres. They enter the parenchyma of liver at the porta hepatis and most accompany the hepatic arteries and bile ducts. A very few may run directly within the liver parenchyma. The capsule is supplied by some fine branches of the lower intercostal nerves, which also supply the parietal peritoneum, particularly in the area of the ‘bare area' and superior surface: distension or disruption of the liver capsule causes quite well localized sharp pain.

Hepatic plexus

The hepatic plexus is one of the largest derivative of the coeliac plexus and receives branches from the anterior and posterior vagi. It usually accompanies the hepatic artery and portal vein and their branches into the liver, where its fibres run close to the branches of the vessels, supplying vasomotor

fibres to the hepatic vessels and biliary tree, and innervating the hepatocytes directly. There Multiple fine branches from the plexus supply the common and hepatic bile ducts directly; branches to the gallbladder form a delicate cystic plexus. vagal fibres are motor to the musculature of the gallbladder and bile ducts and inhibitory to the sphincter of the bile duct.

Nerves from the hepatic plexus run with the branches of the common hepatic artery to supply, or contribute to the supply of, foregut derivatives. Branches which run inferiorly from the plexus with the right gastric artery to contribute to the supply of the pylorus, with the gastroduodenal artery and its branches to reach the pylorus and the first part of the duodenum, and with the right gastroepiploic artery to provide a small contribution to the supply the right side of the stomach and the greater curvature. The superior pancreaticoduodenal extension which supplies the descending part of the duodenum, the pancreatic head and the intrapancreatic part of the common bile duct.

Referred pain

Pain arising from the parenchyma of the liver is poorly localized. In common with other structures of foregut origin, pain is referred to the central epigastrium. Stretch of or involvement of the liver capsule by inflammatory or neoplastic processes rapidly produces well-localized pain of a ‘somatic' nature

MICROSTRUCTURE

The liver is an epithelial-mesenchymal outgrowth of the caudal part of the foregut, with which it retains its connection via the biliary tree. The surface of the liver which is facing the peritoneal cavity is covered by a typical serosa,

the visceral peritoneum. Beneath this, and enclosing the whole structure, is a thin (50–100 μm) layer of connective tissue from which extensions pass into the liver as connective tissue septa and trabeculae. Branches of the hepatic artery and hepatic portal vein, together with bile ductules and ducts, run within these connective tissue trabeculae which are termed portal tracts (portal canals). The combination of the two types of vessel and a bile duct is termed a portal triad

The liver parenchyma which consists of a complex network of epithelial cells, supported by connective tissue, and perfused by a rich blood supply from the hepatic portal vein and hepatic artery. The epithelial cells, hepatocytes,which carry out the major metabolic activities of this organ, but additional cell types possess storage, phagocytic and mechanically supportive functions. In the mature liver, hepatocytes are usually arranged mainly in plates (or cords, as seen in two-dimensional sections) that are usually only one cell thick and separated by venous sinusoids which anastomose with each other via gaps in the plates. Until about seven years of age, plates are normally two cells thick.

Bile secreted by the hepatocytes is usually collected in a network of minute tubes (canaliculi). The hepatocytes can be therefore be regarded as exocrine cells, secreting bile into the alimentary tract via the hepatic ducts and bile duct. Their other metabolic functions involve complex biochemical exchanges with the blood.

The fetal liver is one of the major haemopoietic organ: erythrocytes, leukocytes and platelets develop from the mesenchyme covering the sinusoidal endothelium.

Lobulation of the liver

The structural unit of the liver was the lobule: a roughly hexagonal arrangement of plates of hepatocytes, separated by intervening sinusoids which radiate outward from a central vein, with portal triads at the vertices of each hexagon . The central vein considered as tributary of the hepatic vein that drains the tissue. In some species, the classic lobular units are delimited microscopically by distinct connective tissue septa. Mosthy the lobular organization of the human liver is not immediately evident in histological sections: the lobules do not have distinct boundaries, and connective tissue is sparse. The plates do not pass straight to the periphery of a lobule like the spokes of a wheel but run irregularly as they anastomose and branch.

On Detailed studies of human liver, using three-dimensional reconstruction and morphometric analysis combined with histopathological observations, have revealed that a highly ordered arrangement of functional units, the hepatic (portal) acini. Each acinus had a oval mass of tissue, oriented around the afferent vascular system, i.e. a terminal branch of a hepatic arteriole and portal venule, and with its long axis defined by the territory between two adjacent central veins. It includes the hepatic tissue served by these afferent vessels, and is bounded by the territories of other acini.

The acinar definition of hepatic micro-organization has clarified important problems of liver histopathology, especially the development of zones of anoxic damage, glycogen deposition and removal, and of toxic trauma, which are all related to the direction of blood flow and thus tend to follow the acinar pattern. There were also real metabolic differences between hepatocytes within the acini, and they have been divided into three zones: zone 1 (periportal) is nearest to the terminal branches of afferent vessels; zone 2 is the intermediate zone; and zone 3 is the area closest to the central venous drainage.

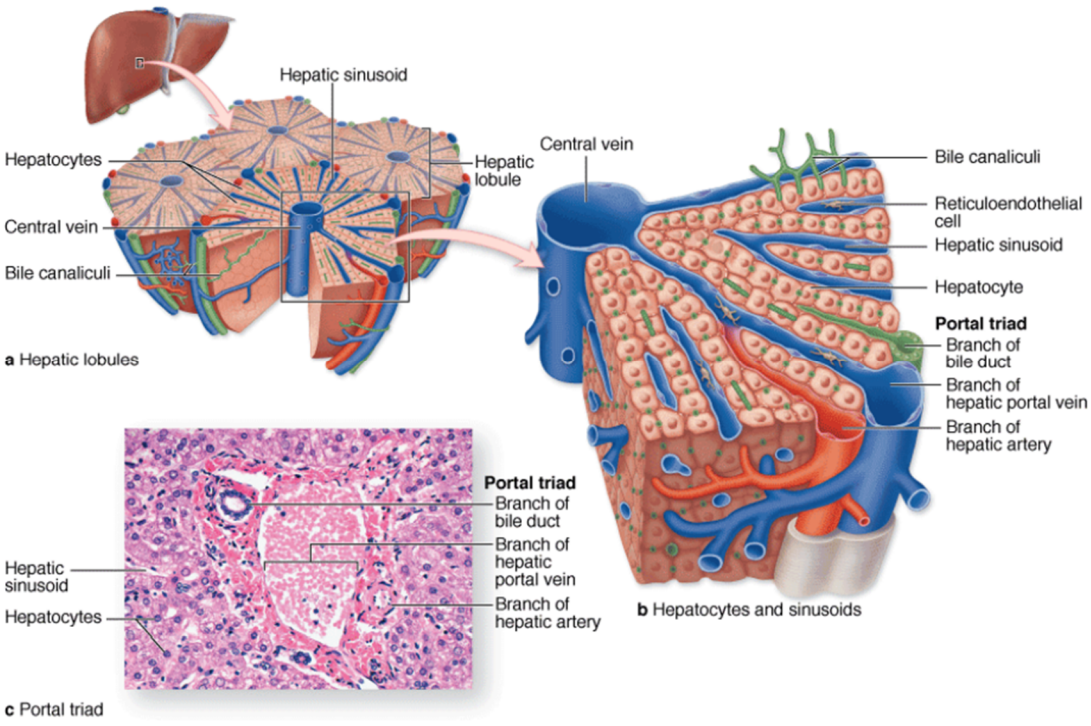


Figure : 11

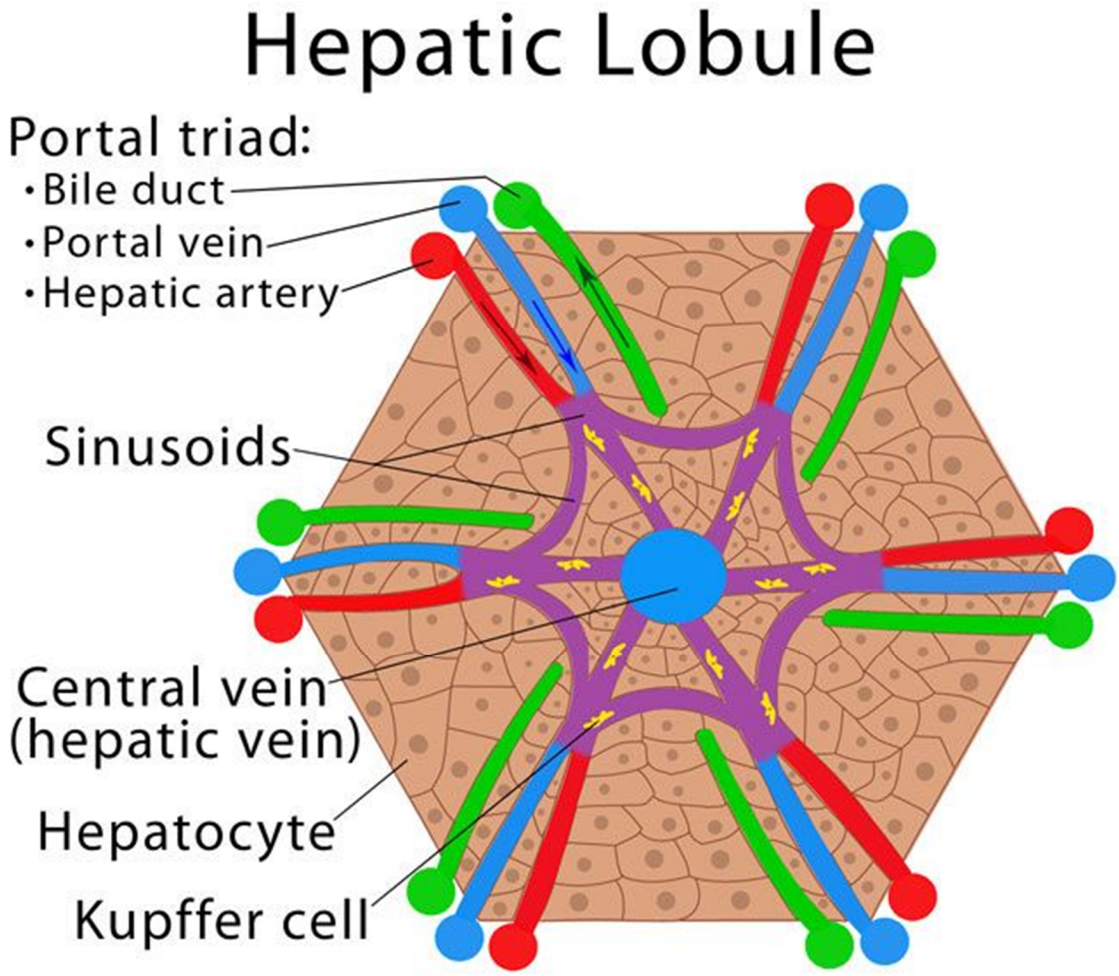


Figure : 12

Blood supply

Preterminal hepatic arterioles which lie in the portal canals branch to convey arterial blood to the sinusoids by several routes. The main course is via a fine capillary plexus which drains to branches of the portal veins. A few arterial blood passes directly to the hepatic sinusoids, bypassing these capillary plexuses, but this represents only a small part of the total flow. Sinusoids thus had mixed venous and arterial blood. Central veins from adjacent lobules form interlobular veins which unite as hepatic veins and drain to the inferior vena cava. Hepatic veins draining the tissue run quite separately with respect to the portal triad system, freely crossing the boundaries of triad territories

Hepatic plates (cords)

The endothelial linings of the sinusoids which are separated from hepatocytes forming the hepatic plates by a narrow gap, the perisinusoidal space of Disse, which is normally about 0.2–0.5 μm wide, but which distends in anoxic conditions. The space contains fine collagen fibres (mostly type III, with some types I and IV), the microvilli of adjacent hepatocytes, and occasional non-myelinated nerve terminals. There is no basal lamina within the space of Disse.

Very small bile canaliculi form nets with polygonal meshes in the hepatic plates. Each polygonal hepatocyte is surrounded by canaliculi (see below) except on the surfaces, at least two per cell, that face sinusoids. Hepatic plates thus enclose a group of canaliculi that pass to the lobular periphery, where they join to form narrow intralobular ductules (terminal ductules or the canals of Hering) lined by squamous or cuboidal epithelium. The intralobular ductules which enter bile ductules, lined by cuboidal or columnar cells, in the portal canals. The flow of bile is thus towards the periphery of lobules, in the opposite direction to the blood flow, which is centripetal.

Cells of the liver

Cells of the liver include hepatocytes, hepatic stellate cells (also known as perisinusoidal lipocytes, or Ito cells), sinusoidal endothelial cells, macrophages (Kupffer cells), the cells of the biliary tree (cuboidal to columnar epithelium) and connective tissue cells of the capsule and portal tracts

Hepatocytes

Majority of about 80% of the liver volume and 60% of its cell number are formed by hepatocytes (parenchymal cells) (Fig. 68.17). They are polyhedral, with 5–12 sides and are from 20 to 30 μm across. The nuclei are round, euchromatic and often tetraploid, polyploid or multiple, with two or more in each cell. Hepatocyte cytoplasm typically contains a considerable amount of rough and smooth endoplasmic reticulum, many mitochondria, lysosomes and well-developed Golgi apparatus, which are all features that indicate a high metabolic activity. Glycogen granules and lipid vacuoles are usually prominent. Numerous large peroxisomes and vacuoles containing enzymes, e.g. urease in distinctive crystalline forms, indicate the complex metabolism of these cells. Their role in iron metabolism is reflected by the presence of storage vacuoles containing crystals of ferritin and haemosiderin.

The surfaces of hepatocytes which are facing the sinusoids exhibit numerous microvilli, approximately 0.5 μm long, creating a large area of membrane, 70% of the hepatocyte surface, exposed to blood plasma. hepatocytes are linked by numerous gap junctions and desmosomes. Lateral plasma membranes of the adjacent hepatocytes form microscopic channels, the bile canaliculi, which are specialized regions of intercellular space which are formed by apposing grooves in hepatocyte plasma membranes, sealed from extraneous interstitial space by tight junctions.a lot membrane-bound exocytotic vesicles cluster near the lumen of the canaliculi because the secretion of bile components is targeted to the canalicular plasma membrane.

These canaliculi which form the origins of the biliary tree and their tight junctions prevent bile from entering interstitial fluid or blood plasma: this is the blood–bile barrier.

Hepatic stellate cells

Hepatic stellate cells are very few compared to that of hepatocytes. They are irregular in outline and lie within the hepatic plates, between the bases of hepatocytes. They are considered to be mesenchymal in origin and are characterized by numerous cytoplasmic lipid droplets. These cells secrete most of the intralobular matrix components, including collagen type III (reticular) fibres. They store the fat-soluble vitamin A in their lipid droplets and are a significant source of growth factors active in liver homeostasis and regeneration. Hepatic stellate cells usually plays a major role in pathological processes. In response to liver damage, they become activated and predominantly myofibroblast-like. They are usually responsible for the replacement of toxically damaged hepatocytes with collagenous scar tissue, a process called hepatic fibrosis, that is seen initially in zone 3, around central veins. Fibrosis can progress to cirrhosis, where the parenchymal architecture and pattern of blood flow are destroyed, with major systemic consequences.

Sinusoidal endothelial cells

Hepatic venous sinusoids are usually wider than blood capillaries and are lined by a thin but highly fenestrated endothelium that lacks a basal lamina. The endothelial cells which are typically flattened, each with a central nucleus and joined to each other by junctional complexes. The fenestrae are grouped in

clusters with a mean diameter of 100 nm, allowing plasma direct access to the basal plasma membranes of hepatocytes. Their cytoplasm contains numerous typical transcytotic vesicles.

Kupffer cells

Kupffer cells are hepatic macrophages derived from circulating blood monocytes and originate in the bone marrow. They are usually long-term hepatic residents and lie within the sinusoidal lumen attached to the endothelial surface. Kupffer cells had a irregular in shape, and have long processes that extend into the sinusoidal lumen. They form a major part of the mononuclear phagocyte system which is responsible for removing cellular and microbial debris from the circulation, and for secreting cytokines involved in defence. They can remove aged and damaged red cells from the hepatic circulation, a function normally shared with the spleen, but fulfilled entirely by the liver after splenectomy

Functions of liver

Liver largest organ and one of the vital organ of the body Metabolic functions – liver is the organ where maximum metabolic reactions such as metabolism of carbohydrates ,proteins fats vitamins and many hormones are carried out Storage functions – substances like glycogen ,aminoacids ,iron ,folic acids and vitamins A,B12,and D are stored in liver

Synthetic functions glucose by gluconeogenesis,plasma proteins ,clotting factors complement factors and hormone binding proteins

Secretion of bile –secrete bile contains bile salts bile pigments ,cholesterol,fatty acids and lecithin .bile salts are required for absorption and digestion of fat

Excretory function-excretes cholesterol ,bile pigments heavy metals ,toxins and bacteria

Heat production –due to metabolic reaction large amount of heat is produced in the

Hematopoietc function-synthesis of haemoglobin

Haemolytic function –senile RBC destroyed by the liver by kupffer cells Inactivation of hormones and drugs –catabolises growth hormone ,parathomone, cortisol,insulin,glucagon and estrogen .also inactivates fat soluble drugs

DEFENSIVE AND DETOXIFICATION

FUNCTIONS

Reticuloendothelial cells (Kupffer cells) of the liver play an important role in the defense of the body. Liver is also involved in the detoxification of the foreign bodies.

i. Foreign bodies such as bacteria or antigens are swallowed and digested by reticuloendothelial cells of liver by means of **phagocytosis.**

ii. Reticuloendothelial cells of liver also produce substances like interleukins and tumor necrosis factors, which activate the immune system of the body .

iii. Liver cells are involved in the removal of toxic property of various harmful substances. Removal of toxic property of the harmful agent is known as detoxification.

Detoxification in liver occurs in two ways:

a. Total destruction of the substances by means of metabolic degradation.

b. Conversion of toxic substances into nontoxic materials by means of conjugation with glu cu ronic acid or sulfates.

Liver Function Tests

Liver function tests are frequently used to refer to measurement of the levels of a group of serum markers for evaluation of liver dysfunction. Most commonly, levels of aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (AP), γ-glutamyltranspeptidase (GGT), and bilirubin are included in this panel. This term is a misnomer, because most of these tests measure not liver function but rather

cell damage. Most accurate measurement of the liver's synthetic function is provided by serum albumin levels and prothrombin time (PT).Eventhough measuring liver enzyme levels is important in the assessment of a patient's liver disease, these test results can be nonspecific. Therefore , evaluation of patients with suspected liver disease should always involve careful interpretation of abnormalities in these liver test results in the context of a thorough history and physical examination. The approach for evaluating abnormal laboratory values also can be simplified by categorizing the type of abnormality that predominates (hepatocellular damage, abnormal synthetic

function, or cholestasis hepatocyte –function test-AST,ALT Synthetic function test PT-INR,factorV,VII,albumin and bilirubin Biliary canalicular function – ALP,5'-nucleotide,GGT

OTHER INVESTIGATION FOR LIVER

1. USG
2. abdomen, angiography,
3. CT Scan,
4. ERCP,
5. MRI,
6. liver biopsy and laparoscopy and laparoscopic US

INFECTIOUS DISEASES

Pathogenesis

The liver is exposed to portal venous bacterial loads on a regular basis and clears this bacterial loads without problem in the usual circumstances

The development of a liver abscess usually occurs when the inoculum of bacteria , irrespective of the route of exposure if it exceeds the livers ability to clear it .This results in the tissue invasion ,and the formation of abscess[1] after neutrophil infiltration

The potential routes of hepatic exposure to bacteria follow

1. Biliary route
2. Portal vein route
3. Hepatic artery route
4. Direct extension from near by foci
5. Trauma

1.Biliary route

Biliary tree infections are is the most common identifiable cause of hepatic abscess[2] . As a result of biliary obstruction there is bile stasis with the potential for subsequent bacterial infecton.,colonisation and which ascends into liver

This process is called as ascending suppurative cholangitis[3] .This nature of biliary obstruction is usually seen in malignancy or stone disease In Asian countries ,stones in the hepatic ductal system and cholangitis are usually the common cause , but in the western world ,malignancy is the predominant factor. Biliary –enteric anastomosis has also been associated with liver abscess formation ,likely due to continuous exposure of the biliary tree to enteric organisms.

2.Portal venous route

The portal venous system drains the major part of the gastrointestinal tract,and any infections which affects the GIT can result in pyelophlebitis with exposure of the liver to large amount of bacteria .The most common cause of pyelophlebitis are the following appendicitis,diverticulitis,inflammatory bowel disease ,pelvic inflammatory disease ,hollow viscus perforation. Liver abscess are also seen in patients with colorectal malignancy

3.Hepatic artery route

Any systemic infection like pneumonia ,endocarditis ,osteomylelitis which can result in bacteremia and infection of the liver occur through the hepatic artery route. Liver abscess from systemic infections occurs as a result of an altered immune response ,such as in those patient with malignancy

,acquired immunodeficiency syndrome ,or disorders of granulocytic function. Childrens with chronic granulomatous diseases are also susceptible to a lesser extent.

4.Direct extension

Hepatic abscess[4] can occur as a result of direct extension of the infective process from other foci.such as sub diaphragmatic abscess ,suppurative cholecystitis ,perinephric abscess and perforation of hollow viscera directly into the liver.

5.Trauma

As a result of Penetrating injury and blunt injury there occur an area of necrotic liver or intrahepatic hematoma ,which later develop into an abscess .Bacteria may have been introduced from the trauma ,or the affected area may be seeded from systemic bacteremia. Liver abscess associated with trauma mostly presents later ,upto weeks after injury .Iatrogenic hepatic necrosis occurs as result hepatic artery embolization or thermal ablative procedures ,can be complicated by abscess.

6.Cryptogenic

In recent case series the cryptogenic abscess are frequently found. The possible theories for this increase in cryptogenic liver abscess are resolved infective foci at the time of presentation ,underdiagnosed abdominal pathology and other factors such as malignancy or diabetes rendering the liver more susceptible to transient hepatic artery or portal vein bacteremias.

Predisposing factors

Pyogenic liver abscess[5] occur more seen in adults associated comorbid conditions like diabetes, pancreatitis,ulcer diseases. Solid organ tumour such as lymphoma and leukemia are present in about 17-36% of patients. These may be due to uncontrolled use of chemotherapy and steroids in such patients.

Host defense abnormalities and immune disorders are most common cause for pyogenic liver abscess in children.complement deficiencies ,chronic granulomatous diseaseand leukemia and other malignancies make the children at increased risk for liver abscess.

Pathology and microbiology

In 75% of cases liver abscess affect the right lobe due to its preferential laminar flow. Only 20 % cases let lobe involved and 5% case it's the caudate lobe . 50% cases its solitary. The size of hepatic abscess may vary from less than1mm to few to several centimetre in diameter andwhich can be multiloculated or uniloculated . the surrounding inflammation which is reason for its adhesion to surrounding organs.

Earlier there were cases of sterile abscess which may occur due to inadequate culture techniques and antibiotic administration before getting in to a diagnosis. Abscess which occur from pyelonephritis and cholangitis are polymicrobial ,mostly by the gram-negative rods. Systemic infection are usually cause infection with single organism.

In 40% of cases the infections are polymicrobial..solitary abscess are caused by polymicrobial .Anaerobes are the most common in about 40-60%

of the time .The most common bacteria that cultured are E-coli and kleb. pneumoniae other organism most commonly found are staph aureus ,enterococcus, strep. viridans and bacteroids species klebsiella which are most commonly associated with gas forming abscess . less common organism are pseudomonas ,enterobacter , proteus, citrobacter, serratia, microaerophilic streptococci, fusobacterium, clostridium 50-60% cases uncommon anaerobes .chemotherapy and immunosuppression are associated with fungal and mycobacterial abscess

Clinical feature

Fever ,jaundice ,weightloss,pain nausea and vomiting ,malaise ,chills ,anorexia ,cough and pleurisy ,pruritus and diarrhoea are most commonly seen clinical features.

Physical examination

Right upper quadrant tenderness ,hepatomegaly ,jaundice ,right upper quadrant mass ,ascites ,pleural effusion are the finding we come across in pyogenic abscess

Laboratory investigations

Increased alkaline phosphatase TC count elevated ,albumin <3g/dl,haematocrit <36%,bilirubin >2g/dl

Imaging studies

Radiological imaging plays a pivot role in diagnosis

Chest X-ray

Chest x-ray are abnormal in 50% of the cases ,finding correlates with sub diaphragmatic pathology such as pleural effusion ,an raised right hemidiaphragm or atelectasis sometime there will be left sided findings in the case of an abscess involving left liver

X-ray abdomen erect

Plain abdominal x-rays can be helpful only in few cases only. Finding seen as portal venous gas or air fluid levels. Plain abdominal x-ray demonstrating an abnormal collection of air in the right upper quadrant consistent with pyogenic hepatic abscess(white arrow)

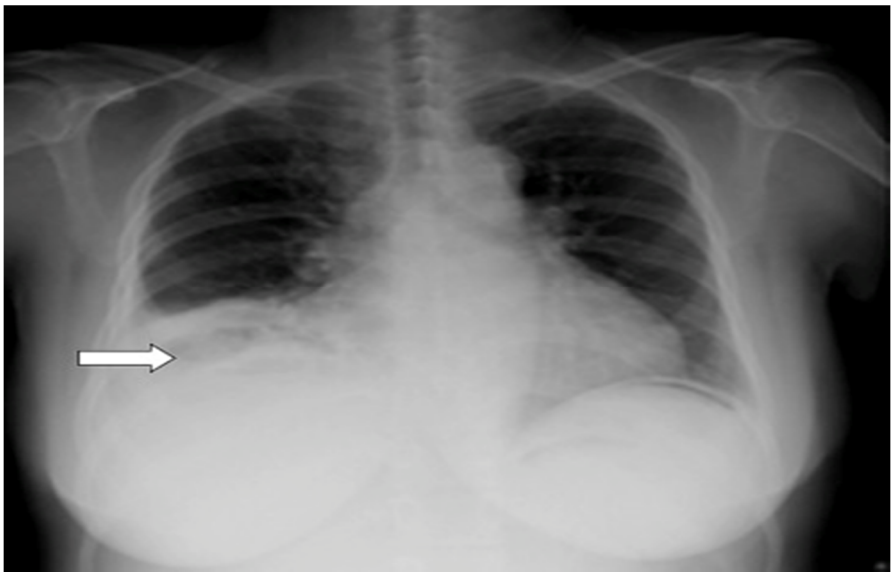


Figure : 13

USG and CT scan are mainstays in diagnosis of liver abscess

Ultra sonogram abdomen

USG usually demonstrates

1) Oval or round area which is less echogenic compared to liver
2) Distinguish solid from cystic lesion

The sensitivity is about 80-90%

CT abdomen plain and contrast

CT demonstrates findings similar to ultrasound and lesions are of lower attenuation compared to surrounding liver parenchyma .High quality CT can identify very small abscess of even 0.5cm and which can easily identify multiple small abscess .it cannot be limited by air or ribs .the abscess wall usually show an intense enhancement on contrast –enhanced CT .the sensitivity of CT in diagnosing liver abscess is 95-100%

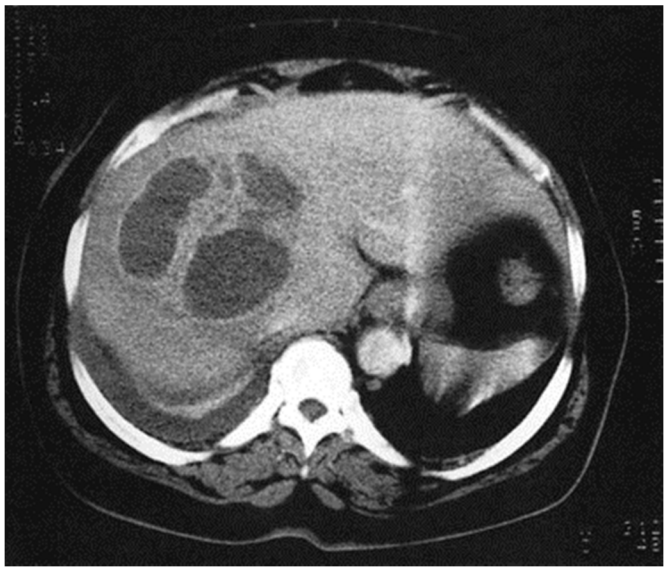


Figure : 14

Both CT and USG are useful in diagnosing other abdominal pathology,and help us to distinguish from other inflammatory pathology like appendicitis and diverticulitis.

Differential diagnosis

1) Amoebic liver abscess
2) Echinococcal cyst

Feature of amoebic liver abscess versus pyogenic liver abscess

Table : 2

Clinical features	**Amoebic[6]**	**Pyogenic**
Age	20-40	>50
Male to female ratio	10:1	1.5:1
Solitary vs multiple	Solitary 80%	Solitary 50%
Location	Right lobe	Right lobe
Diabetes	Uncommon	More common
Travel in endemic area	Yes	No
Alcohol use	Common	Common
Jaundice	Uncommon	Common
Elevated bilirubin	Uncommon	Common
Elevated alkaline phosphatase	Common	Common
Positive blood culture	No	Yes
Positive amoebic serology	Yes	No

Amoebic liver abscess 50% are solitary.

Treatment

Earlier it was a fatal. It was not until the classic review by ochsner and DeBakey in 1938 that routine surgical drainage was intoduced and there was a dramatic reductions in mortality. Until 1980s the open drainage and antibotics

came into play then only less invasive percutaneous drainage techniques with antibiotics was employed. Laparotomy was reserved for failed cases.

When the diagnosis of pyogenic abscess was suspected ,broad spectrum IV antibiotics ahould be started as soon as possible to control the bacterial load and the associated complications with liver abscess.

Patients who are at risk[7] for amoebics infection for them amoebics serologies should be sent immediately .until culture identified the causative organism broad spectrum antibiotics covering all gram negative ,gram positive and anaerobes should be started empirically.

Medical management

Combinations such as ampicillin ,aminoglycoside and metronidazole or a third generation cephalosporins with metronidazole should be started. The optimum duration of antibiotic treatment not correctly seen in any literatures and must be individualised depending on the success of the drainage procedure. Antibiotic are continued until the ongoing infection settles down such as fever with chills or leucocytosis, but usually prescribed for 2 or more weeks

Percutaneous drainage procedure

Earlier percutaneous catheter drainage has been treatment of choice .success rate was about 69-90%

Advantages

1) The simplicity of treatment
2) Prevents general anaesthesia

3) Prevents laparotomy

Contraindications

1) Ascites
2) Coagulopathy
3) Proximity to vital structures

Despite of comparison between the percutaneous drainage with surgical treatment overall mortality and morbidity are the same

In general ,surgery is reserved for patients who needs the treatment for primary pathology[8] or for those who have failed percutaneous techniques

Percutaneous aspiration

Percutaneous aspiration[9] is a technique in which there is placement of a drain. Success rate is about 60-90% when compared to percutaneous catheter drainage[10] .usually most of them needs ,more than one aspiration is required and one –fourth of patients require more than three aspirations[11] as a routine. In randomized trial success rate in those patient undergone aspiration is 60% and 100% in catheter drainage. In general catheter drainage is the treatment of choice in most of the cases

Open drainage

For open drainage these factors should be needed

1) Recurrent abscess
2) Thick pus
3) Multiloculated
4) Left lobe abscess

5) Multiple abscess

6) Ruptured abscess

7) Failed medical and image guided aspiration

Liver resection

Very rare for liver abscess is indicated when an infected hepatic malignancy ,hepatolithiasis or intrahepatic biliary stricture . if major liver parenchyma is destroyed ,those patients may require resection.

Outcome

Mortality due to pyogenic liver abscess has been reduced dramatically during past 6 decades. Pyogenic abscess was uniformly fatal before there was proper protocol for treatment. With the routine use of surgical drainage and use of IV antibiotics mortality reduced drastically to about 50%. Since the 1980s mortality has been reported from 10-20% and series from the 1990s now routinely demonstrate a mortality rate of less than 10%.

Factors predictive of a poor outcome

- The presence of malignancy
- Factors associated with malignancy(jaundice ,elevated LFTs)
- Signs of sepsis
- Hypoalbuminemia
- Signs of severe infection
- Abscess rupture
- Bacteremia
- Shock

Amoebic liver abscess

Epidemiology

Amoebic liver abscess most commonly seen in males aged 20-40 years ,in most cases with a history of travel to an endemic area.

Poverty and cramped living conditions is the reason for higher rates of infection . a very high male predominance of greater than 10:1 is seen .heavy alcohol drinking commonly reported and make the liver more susceptible to amoebic infection

Patient with altered host defence mechanism also appear to be at higher risk for infection and have higher mortality rate. Patients with amoebic liver abscess with no history of travel is commonly seen in patients with immunosuppression such as human immunodeficiency virus infection ,malnutrition ,chronic infection ,or chronic steroid use.

Pathogenesis

Entamoeba histolytica is a protozoa that exists as a trophozoite or as a cyst

All other species in the genus entamoeba are found to be non pathogenic and not all strains of entamoeba family is causing infection. Intake of cysts of entamoeba occurs through feco –oral route . humans are principal host and the main source of infection is human contact with a cyst passing carrier.

Contaminated water and vegetables may also act as source of human infection . once ingested ,the cyst are not degraded in the stomach and pass to

the intestine where the trophozoite is released and passed on to colon .in the colon ,the trophozoite can invade mucosa resulting in disease.

Those trophozoite which reach the liver through the portal venous system. There is no evidence to say that trophozoite passing through lymphatics. As implies by its name E.histolytica trophozoites have the capacity to destroy tissues through a complex set of events ,including cell adherence ,cell activation and subsequent release of multiple enzymes resulting in necrosis. The major mechanism is probably enzymatic cellular hydrolysis. Amoebic liver abscess occurs as a result of a progressing ,localized hepatic necrosis resulting in a cavity containing acellular proteinaceous debris which is surrounded by a rim of invasive amoebic trophozoites. Early development of an amoebic liver abscess with blood picture of polymorphonuclear leucocytosis which are then destroyed by trophozoites

Antiamoebic antibodies develop rapidly in patients with invasive disease or amoebic hepatic abscess. Secretory immunoglobulin A antibodies have been shown invitro to inhibit adherence to colonic epithelium ;however ,the development of these antibodies does not further stop the progression of the disease. Interestingly children who lack antibodies IgG having innate resistance to infection saying an alternative immune mediated response. There is now evidence the cell mediated T-helper response is probably the major mechanism of resistance.

Pathology

Amoebic liver abscess occurs as a result of liquefaction necrosis of the liver ,producing a cavity filled with blood and liquefied liver tissue

The appearance of this fluid is typically defined as **anchovy sauce** and fluid is odourless unless secondary bacterial infection occur.

The progressiveliver necrosis continues upto glissons capsule,because the capsule is resistant to hydrolysis by the amoeba and thus amoebic abscess tends to abut liver capsule. Because of glissons capsule ,the cavity is mostly crisscrossed by portal triads protected by this peritoneal sheath. Early on ,the cavity which formed is ill defined with no real fibrous response around the edges ,but a chronic abscess can ultimately develop a fibrous capsule and which tend to calcify .like pyogenic abscess amoebic liver abscess tend to occur in the right lobe.

Most common location

Most common site is right posterosuperior segment

Clinical features

- It is mostly seen in males (20:1) may be after a week of an attack of amoebic dysentery or many months after the attack
- Usually Present with fever weight loss chills rigor ,non productive cough ,shoulder pain
- Pain in the right hypochondrium is the classical sign
- Soft ,tender and smooth liver with increased span
- Inter costal tenderness

- Right sided pleural effusion
- Mild jaundice is may be sen in cirrhotic and multiple abscess
- Tenderness , rigidity and skin edema in right quadrant
- In chronic abscess smooth ,firm/hard non tender liver may be palpable

Amoebic liver abscess may be:-

- Acute – present with high fever ,chills ,rigor,tender liver with intercostal tenderness
- Chronic –present with firm/hard ,smooth ,nontender palpable liver without acute features

Features may be of –

- Systemic –presents with fever,chills and rigor ,there will be loss of appetite with reduced weight and sometimes there will be jaundice
- Abdominal –usually present with pain and tenderness , there will be localized guarding and sometimes localised rigidity, mass in the right upper abdomen splenomegaly and abdominal wall oedema
- Thoracic –there will be dry coughwith associated chest pain in the right lower part ,right shoulder pain pleural effusion and intercostal tenderness
- Features of complication –rupture /infection/septicaemia/liver failure

Differential diagnosis

For Acute type

1. Acute cholecystitis
2. Acute presentation of HCC

3. HCC due to haemorrhage or necrosis

Sub phrenic abscess

Chronic amoebic liver abscess mimic hepatoma in every aspect

Investigations

- Total count raised. Blood picture of mild to moderate leucocytosis
- LFT may shows an altered bilirubin and albumin level
- Prothrombin time will be widened and if its so inj.vit K is given for 5 days. Even PT is elevated FFP transfusion should be done immediately.
- AST,ALT ,serum alkaline phosphatase levels are altered
- Indirect haemagglutination test (95% positive rate)
- **Chest** xray findings

-raised fixed diaphragm

-pleural effusion

-soft tissue shadow

-Atelectasis

- USG findings
  - Altered echogenicity
  - Size of the lesion
  - Location of abscess in liver
  - Number of abscess present
  - Nature of liver

Abdominal USG with accuracy of about 90% along with typical history and clinical examination

- The USG has peripheral location rounded shape with poor rim and internal echoes

CT scan – both plain and contrast shows raised diaphragm and abscess cavity – it gives an idea about location ,size ,number ,presence of effusion and changes in lung as a result of abscess

Abdominal CT scan help us in differentiating amoebic with pyogenic liver abscess ,with a rim enhancement noted in pyogenic liver abscess.

CT scan of amoebic liver abscess , the lesion is usually located in periphery and round , with a non enhancing rim,but shows peripheral oedema

Figure : 15

- Sigmoidoscopy

  Used to identify active ulcers ,scrappingfrom the ulcer base on mounding show trophzoites

Management

The main drug used in treatment of amoebic liver abscess is metronidazole 750mg tds for 10 days orally which cure the disease in 90% of cases. Clinical improvement is usually occur in within 3 days. If response to metronidazole is poor other agents must be tried .

Emetine hydrochloride used against invasive amoebiasis ,but requires intramuscular injections and will show serious side effects. Another attractive option is chloroquine unfortunately is a less effective agent. After treatment of liver abscess it is recommended that luminal agents such as iodoqinol ,paromomycin and diloxanide furoate are usually given to carriers.

Aspiration

- In case of large abscess and infected aspiration with a wide bore needle under USG guidance
- Previously without USG aspiration is done by inserting a needle in right 6^{th} intercostal space in midaxillary line

In general aspiration is done for diagnostic uncertainty and those with failure of metronidazole therapy or in cases where chance of rupture is high. Abscess greater that 5cm and left liver is thought to be high risk of rupture and aspiration should be considered.

Percutaneous drainage

Under USG guidance a pig tail catheter is introduced into the abscess cavity percutaneouslyfor drainage of the pus. Catheter tube and abscess cavity should be washed and irrigated at regular intervals with normal saline. In certain situations it fails like thick pus .multiloculated and multiple abscess. Procedure may cause bleeding and infection.

Figure : 16

Through the transperitoneal approach abscess cavity is opened ,pus is evacuated. Malecot catheter is placed and brought out through a separate stab incision. The catheter kept in place until the drainage stops completely. Complete stoppage of drain is confirmed by repeat ultrasoundof the abdomen. During discharge ,advice given to stop alcohol ,chloroquine 250mg BD for 10 days and diloxanide 500mg tid for 2 weeks.

Complications of surgery- anaesthetic problem, sometime liver failure ,rarely intraperitoneal abscess formation ,bile leak and biliary peritonitis

Indication for surgery

1. Even after repeated aspiration cavity fills
2. Thick pus
3. Multiloculated abscess
4. Left lobe abscess because of rupture into pericardial cavity
5. Ruptured abscess
6. Caudate lobe abscess
7. Multiple abscess

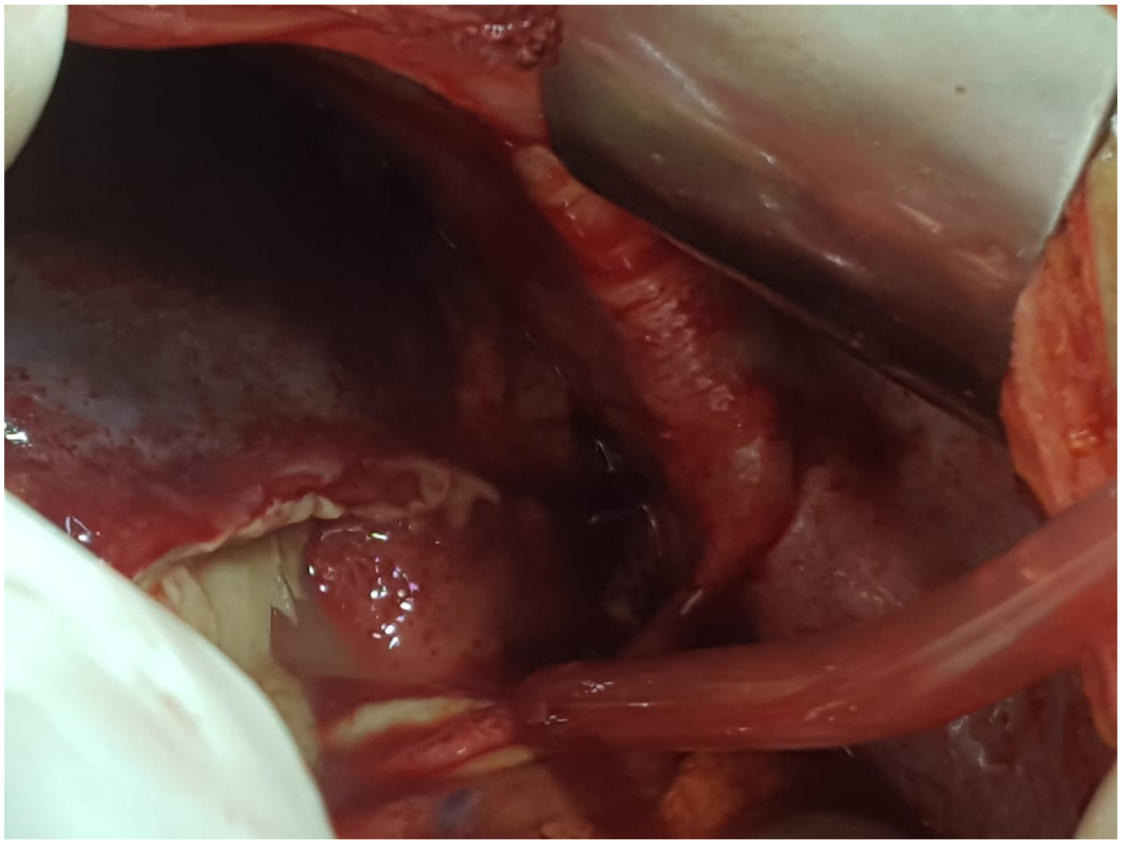
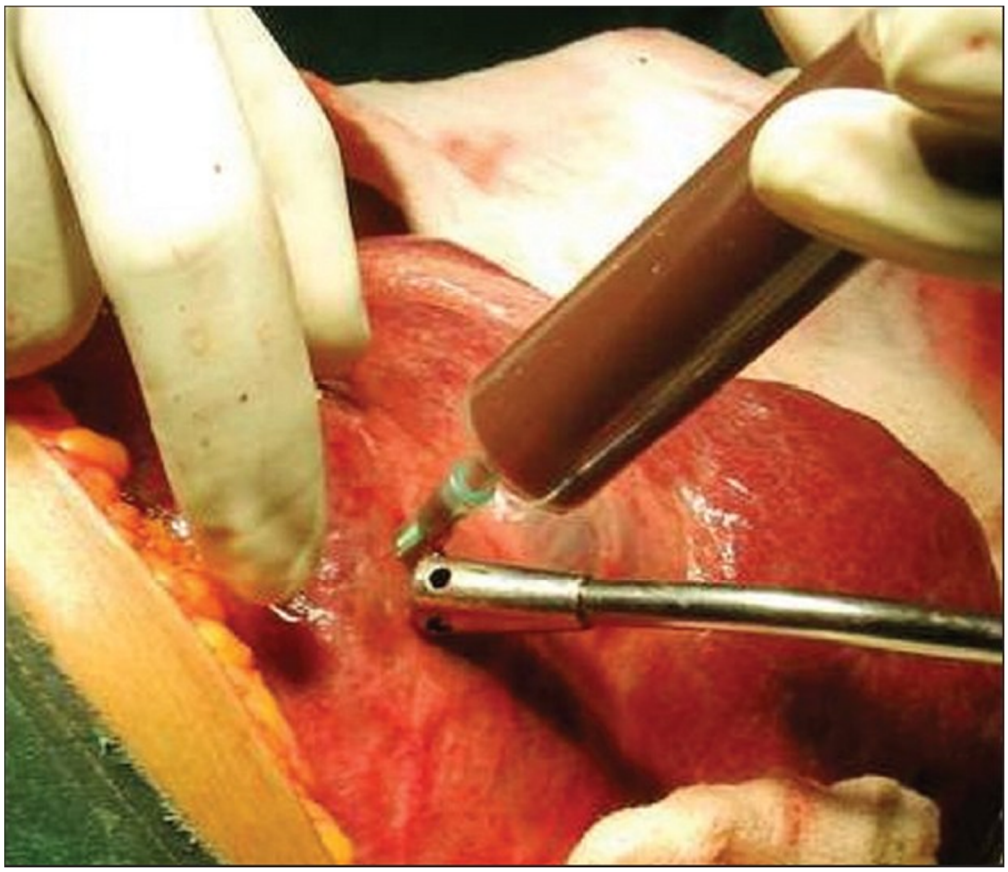
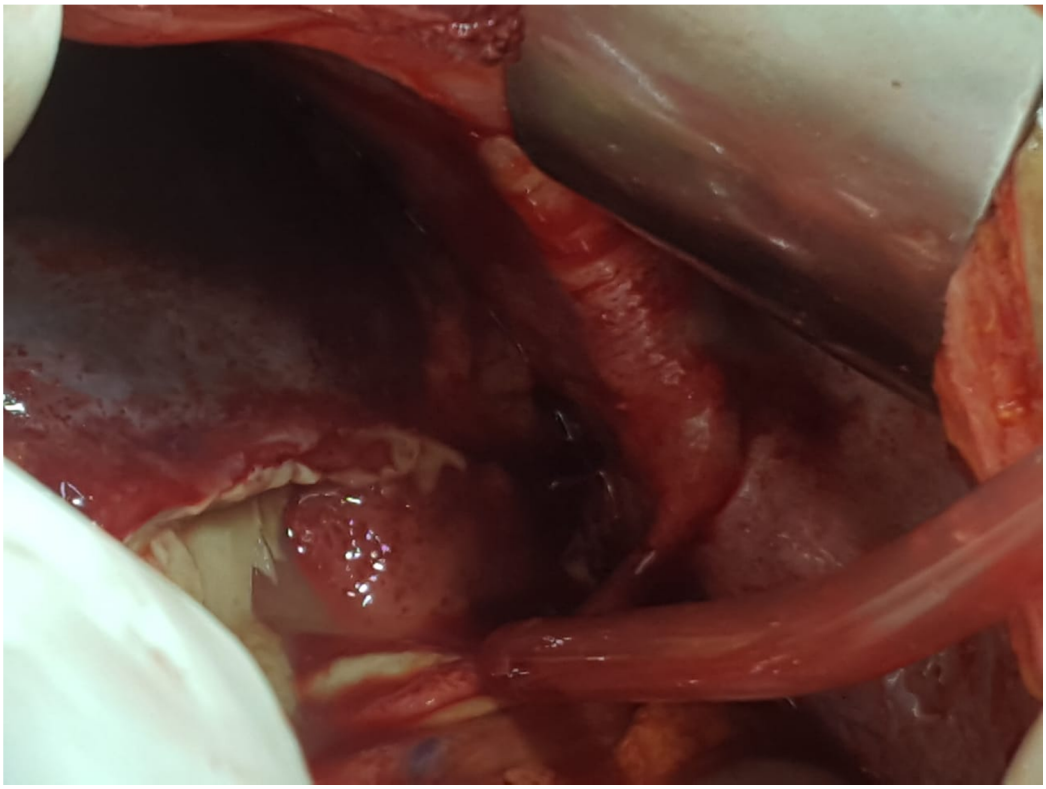


Figure : 17





Outcome

Although amoebic liver abscess respond rapidly to treatment ,sometime ver rarely presents with complications. The most common complication is rupture into peritoneal cavity or pericardium. Size of abscess is common risk factor for rupture ,and the overall incidence for thee liver abscess to rupture is 3 to 17%. Most peritoneal rupture is contained by diaphragm abdominal wall or omentum ,but rupture can fistulise to hollow viscus. A peritoneal rupture,patient presents with abdominal pain ,peritonitis and either as mass or distension. So laparotomy was advocated for this. Rupture into pleural cavity results in large and rapidly accumulated effusion that collapses the lung,which is treated by thoracocentesis. Rarely left sided abscess may rupture into pericardium and can present as cardiac tamponade or asymptomatic pericardial effusion. Other complication include compression of biliary tree and inferior venecava and may also result in development of brain abscess

The mortality for all patients of amoebic liver abscess is about 5% do not get altered by treatment. When an abscess ruptures the mortality rate is raised from 6% to as high as 50%. Factors that indicates poor outcome are elevated LFT ,encephalopathy,hypoalbuminemia ,multiple abscess and abscess volume of greater than 500ml, anemia and diabetes

Although clinical improvement with adequate treatment with antiamoebic agents is a rule ,there will be radiological resolution of abscess. The average time for radiological resolution is 3-9 months may also take years to resolve . Studies have shown that more than 90% of the visible lesion appear radiologically ,but a small percentage of patients are left with a clinically irrelevant residual lesion.

MATERIALS AND METHODS

Study design:

Prospective cohort study

Methodology :

Patients presenting with features of liver abscess and diagnosed radiologically or yet to be diagnosed are included in the study and detailed clinical and laboratory history, treatment as per standard guidelines, factors leading for surgical intervention and subsequent follow-up are carried out and the data will be analysed.

The common factors associated with surgical intervention in cases of liver abscess include

Table : 3

S.NO	FACTORS
1.	Residual / Recurrent abscess
2.	Thick pus
3.	Multi-loculated abscess
4.	Left lobe abscess
5.	Ruptured abscess
6.	Caudate lobe abscess
7.	Multiple abscess

Sample size:

Patients admitted with liver abscess at CMCH from January 2017 – december 2017

Sample size-144

Inclusion criteria :

1. Radiologically diagnosed liver abscess
2. All cases of bacterial and parasitic liver abscess
3. All cases of evolving ,liquefied and ruptured liver abscess with or without peritonitis
4. Past history of liver abscess
5. Al the cases of diagnosed liver abscess referred to CMCH

Exclusion criteria :

1. Liver abscess associated with malignancy
2. Immunocompromised state

RESULTS

Table 4: sex distribution in sample

SEX DISTRIBUTION		
SEX	FREQUENCY	PERCENT
MALE	122	84.7
FEMALE	22	15.3
TOTAL	144	100

The study sample showed a male predominance.

Figure 18: Sex Distribution in Sample

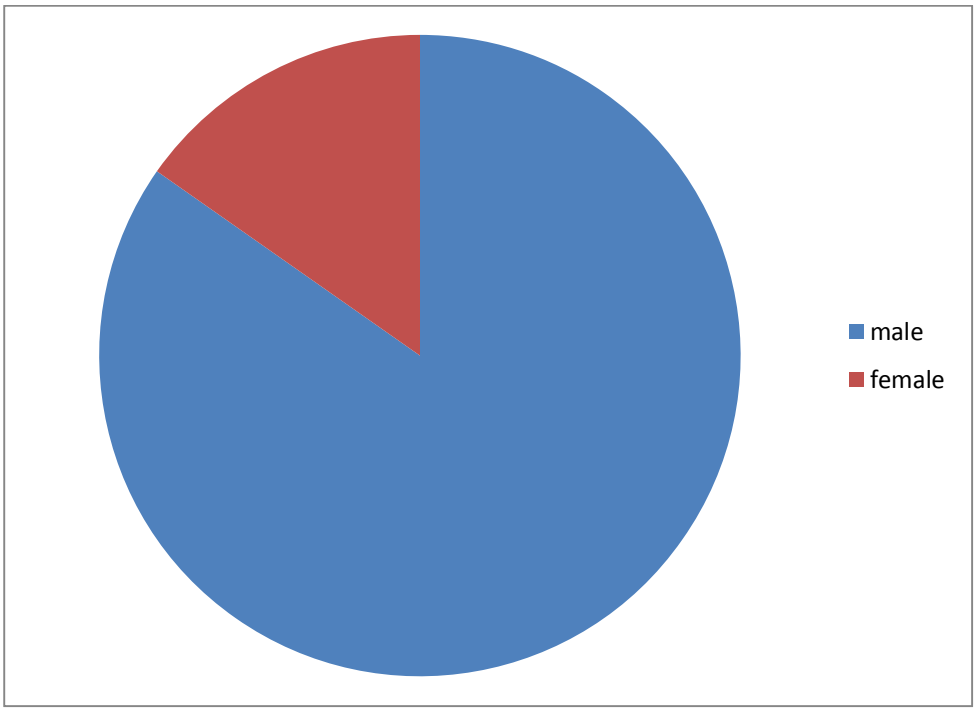


Table 5: Age Distribution In Sample

AGE DISTRIBUTION	
AGE	**NUMBER**
<20	0
21-40	36
41-60	88
>60	20
	144

Predominantly patient belongs to 41-60 age group

Figure 19: age distribution

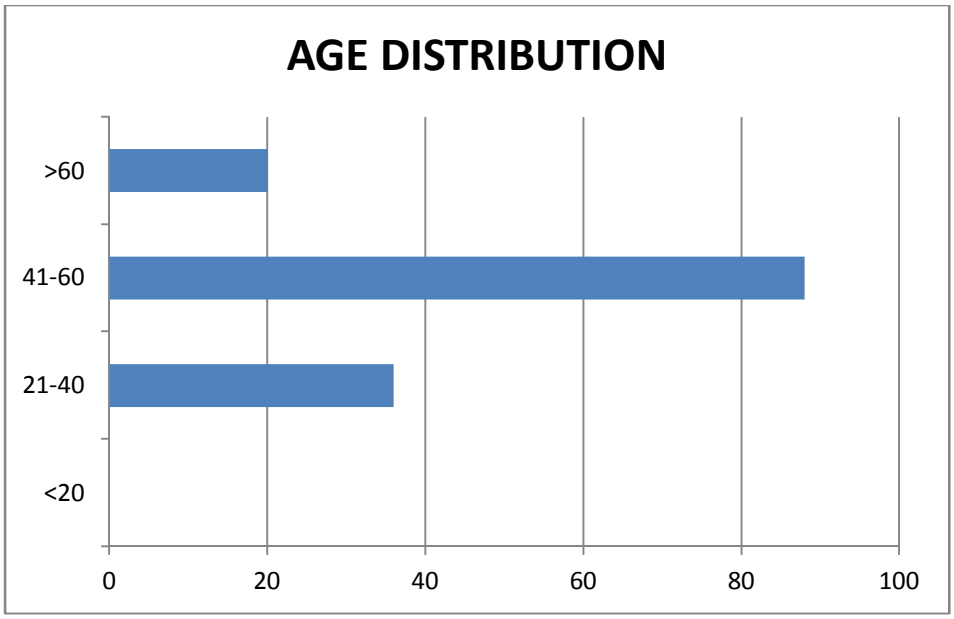


Table 6: Liver lobe involved

LOBE INVOLVED			
MANAGEMENT	**RIGHT**	**LEFT**	**TOTAL**
OPERATIVE	44	32	76
CONSERVATIVE	56	12	68
TOTAL	100	44	144

Figure 20: liver lobe involved

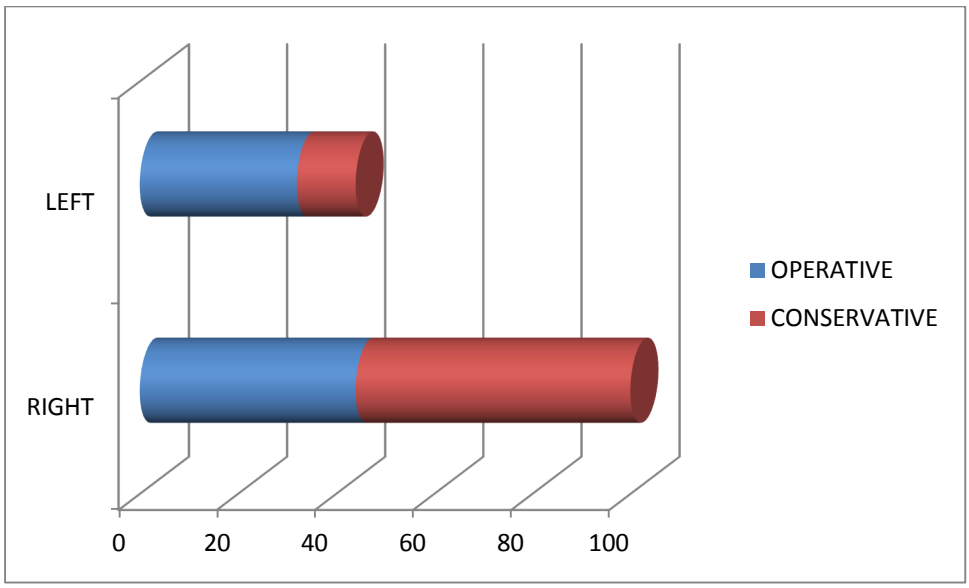


Table 7: character of pus

CHARACTER OF PUS			
MANAGEMENT	**THICK**	**THIN**	**TOTAL**
OPERATIVE	32	44	76
CONSERVATIVE	16	52	68
TOTAL	48	96	144

Figure 21: character of pus

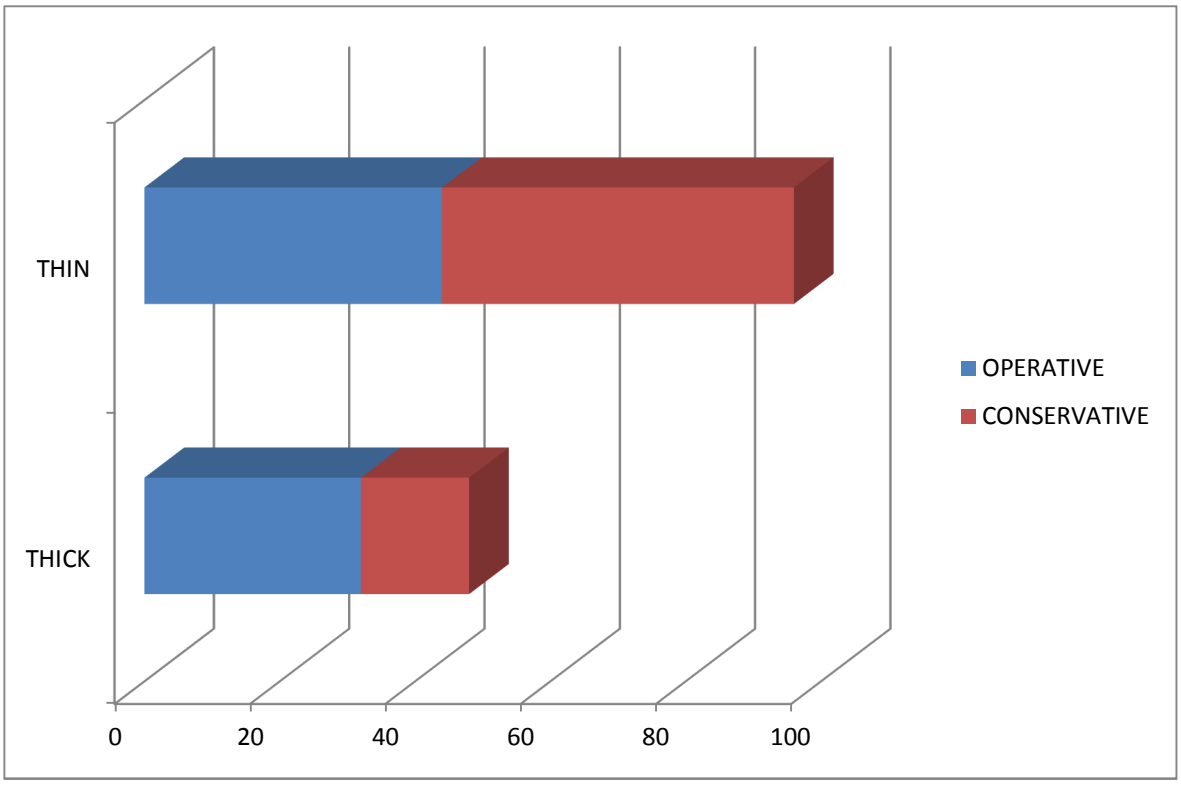


Table 8: Number of loculations in abscess

LOCULATED ABSCESS			
MANAGEMENT	UNILOCULATED	MULTILOCULATED	TOTAL
OPERATIVE	32	44	76
CONSERVATIVE	60	8	68
TOTAL	92	52	144

Figure 22 : number of loculations in abscess

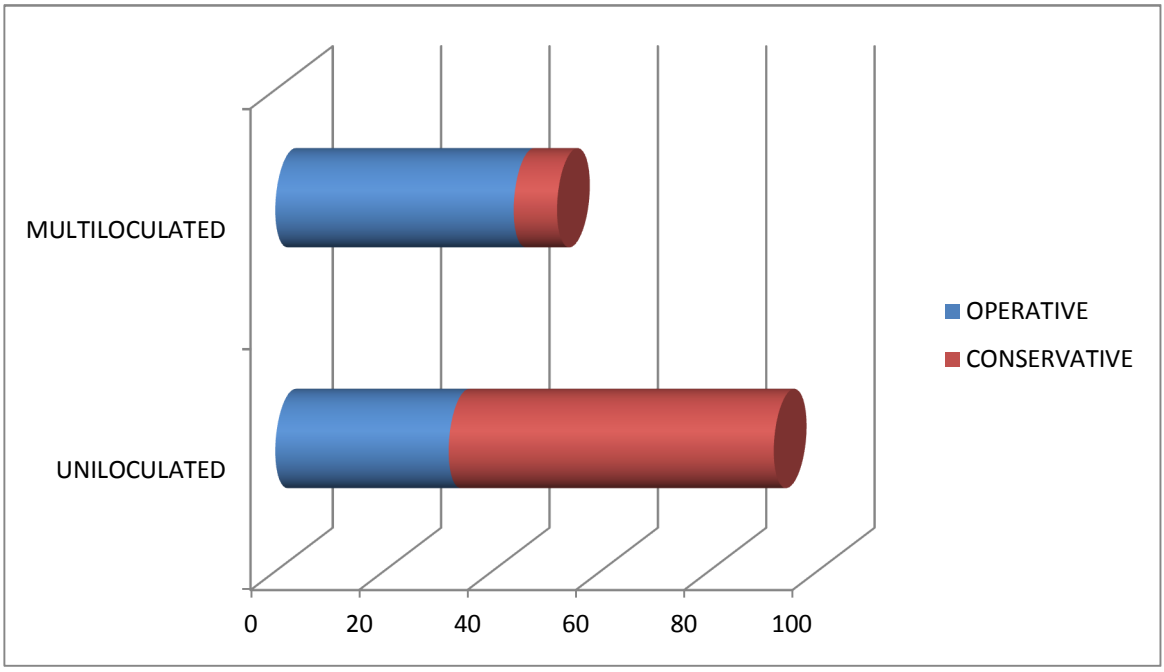


Table 9: number of lesions

NUMBER OF LESIONS			
MANAGEMENT	**SINGLE**	**MULTIPLE**	**TOTAL**
OPERATIVE	40	36	76
CONSERVATIVE	56	12	68
TOTAL	96	48	144

Figure 23: number of lesions

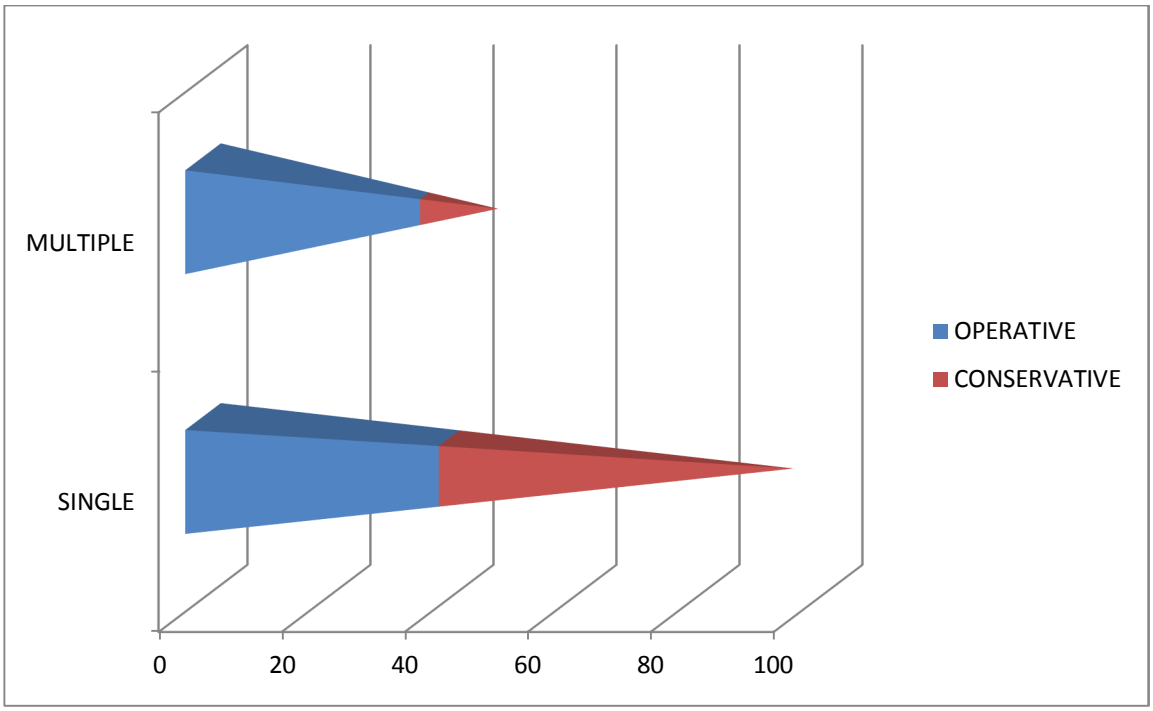


Table 10: primary vs recurrent liver abscess

PRIMARY VS RECURRENT			
MANAGEMENT	**PRIMARY**	**RECURRENT**	**TOTAL**
OPERATIVE	56	20	76
CONSERVATIVE	64	4	68
TOTAL	120	24	144

Figure 24: primary vs recurrent liver abscess

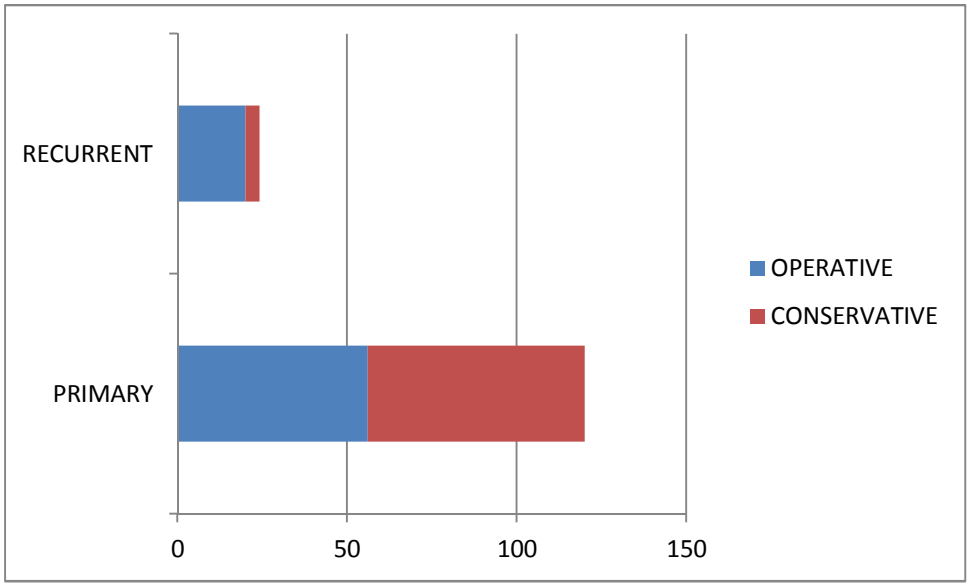
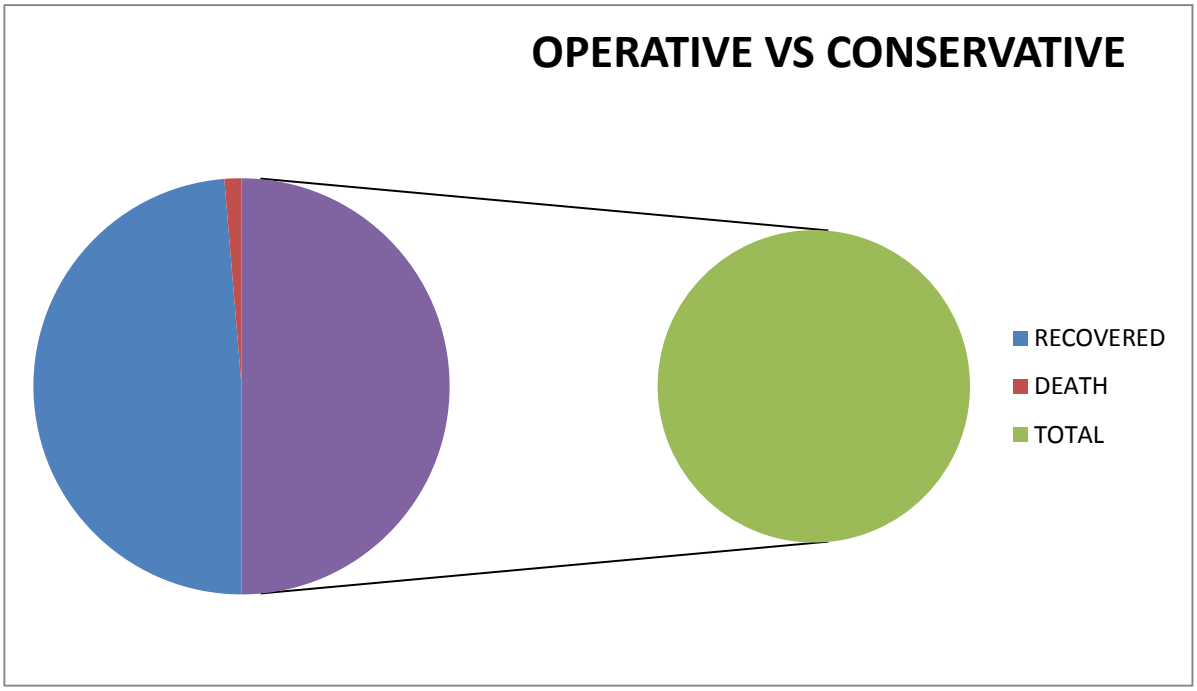


Table 11: Outcome of Liver Abscess Management

OUTCOME			
MANAGEMENT	**RECOVERED**	**DEATH**	**TOTAL**
OPERATIVE	74	2	76
CONSERVATIVE	64	4	68
TOTAL	138	6	144

Figure 25: Outcome of Liver Abscess Management



On computing the observed and expected values of various determinants in the 2x2 table and applying chi-square tests the following results were obtained

1. Among 100 right lobe abscess 44 were treated operatively and 56 conservatively. In 44 left lobe abscess 32 were operated and 12 treated conservatively. On chi-square testing the calculated is 10.09 >6.83 ,hence p value <0.01 ,which shows statistical significance.
2. Among 48 patients with thick abscess 32 were treated operatively and 16 were treated conservatively. In 96 patients with thin abscess 44 treated operatively and 52 treated conservatively. On chi-square testing the calculated value is 5.57 >3.84 ,hence p value <0.05,which shows statistical significance.
3. Among 52 patients who are having multiloculated abscess 44 were treated operatively and 8 treated conservatively. In 92 patients with uniloculated abscess 32 treated operatively and 60 treated conservatively. On chi-square testing the calculated value is 33.1 >10.84 ,hence p value is <0.001 ,which shows statistical significance.
4. Among 48 patient with multiple abscess 36 treated operatively and 12 treated conservatively. In 96 patients with single abscess 40 treated operatively and 56 treated conservatively. On chi-square testing the calculated value is 14.25 >10.84, hence p value <0.001 ,which shows statistical significance

5. Among 24 patients with recurrent abscess 20 treated operatively and 4 treated conservatively. In 129 patients with primary abscess 56 treated operatively and 64 treated conservatively. On chi-square testing the calculated value is 10.79 >6.83,hence p value is <0.01, which shows statistical significance
6. Out of 76 patients operated for liver abscess 2 patients died and rest recovered ;68 patients treated conservatively ,64 recovered and 4 died. On computing in chi-square testing the calculated value is 0.95 <3.84,hence p>0.05 ,so null hypothesis accepted. That is no much statistical significance among outcome in surgical and conservative treatment modalities.

DISCUSSIONS

Liver abscess has multimodal aetiological and risk factors, varied presentation ,management approaches and high recurrence or residual disease

Hence planned to study the various clinical presentation ,management modalities and factors deciding necessity for surgical intervention in liver abscess and outcome

Hence we studied various factor deciding necessity for sutgical intervention in liver abscess. Chi –square test was applied on obtained parameters to arrive for statistical significance

The results showed

1. Among left lobe liver abscess vs right lobe liver abscess treated ,the p value <0.01 shows statistical significance
2. Among thick pus vs thin pus treated ,the p value is <0.05 shows statistical significance
3. Among multilocated abcess vs uniloculated abscess treated ,the p value is <0.001 which shows high statistical significance
4. Among multiple abscess vs single abscess treated ,p value is <0.001 which shows statistical significance.
5. Among recurrent abscess vs primary abscess treated ,p value is <0.01 which shows statistical significance

So these following parameters shows necessity for surgical intervention

1. Recurrent abscess
2. Thick pus
3. Multiloculated abscess
4. Left lobe abscess
5. Caudate lobe abscess
6. Multiple abscess

The calculated value for outcome between operated and conservative management ,here the p value is<0.05 hence there no significance ,the null hypothesis is accepted

CONCLUSION

Various factors demanding for surgical intervention in liver abscess was studied as a prospective study at Coimbatore medical college hospital in department of general surgery for a period of one year. Datas obtained were analysed using chi-square test.

The following parameters shows the necessity for surgical intervention in liver abscess

1. Recurrent abscess
2. Multiloculated abscess
3. Thick pus
4. Multiple abcess
5. Caudate lobe abscess
6. Left lobe abscess

The above parameters showed statistical significance

The calculated value for outcome between operative and conservative management showed no statistical significance, hence null hypothesis accepted.

This indicates necessity for study in large sample size and multicentric studies to arrive for a conclusion to prove statistical significance regarding outcome

SUMMARY

Liver abscess has multimodal aetiological and risk factors, varied presentation ,management approaches and high recurrence or residual disease

Hence planned to study the varying clinical and biochemical presentations of liver abscess, risk factors, management modalities, factors deciding necessity for surgical intervention and outcome.

The prospective study was conducted at Coimbatore medical college for one year duration after obtaining local ethical committee permission

Based on the incidence at this college the sample size calculated as 144 patients. The various factors deciding the necessity of surgical intervention were studied, the following parameters shows the necessity for surgical intervention. They were recurrent abscess, thick abscess, multiple abscess, multiloculated abscess, left lobe abscess.

On applying chi-square test on obtained parameters there was stastistical significance for individual factors.

But the calculated valve for outcome between operated and conservative management does not showed any statistical significance. To arrive for a conclusion regarding statistical significance in outcome it needs study in large sample size and multicentric studies .

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ANNEXURE – I

PROFORMA

Date: Time : Informant:

Referral / direct : Referral slip/summary – enclosed / not enclosed

Resident Sign :_____

Asst Prof. Sign: _____

PRESENTING COMPLAINTS:

1.
2.

HISTORY OF PRESENT ILLNESS:

PAST HISTORY:

COMORBID ILLNESS	PRESENCE	HOW LONG	MEDICATION
Hypertension			
Diabetes mellitus			
Asthma/COPD			
CAHD/cardiac			
Jaundice			
Tuberculosis			
CNS/epileptic			
others			

Previous blood transfusion :

PERSONAL HISTORY :

Diet : Occupation: Marital Status:

Alcohol: Smoking : other addiction:

MENTRUAL HISTORY:

Menarche: Cycles: LMP: Menopause:

OBSTETRIC HISTORY:

Parity : LCB: sterilization:

FAMILY HISTORY:

DRUG HISTORY:

Medications: allergy:

PREVIOUS SURGICAL HISTORY:

GENERAL EXAMINATION: Weight:

Conscious			Cyanosis	
Orientation			Clubbing	
Temperature			Lymphadenopathy	
Pallor			B/L Pedal edema	
Icterus			Hydration	

VITALS:

B.P	
PR	
RR	

SYSTEMIC EXAMINATON:

CVS: HEART SOUNDS MURMUR:

RS: TRACHEA: BREATH SOUNDS:

ADDED SOUNDS:

CNS: HMF: PNS: ANS:

INDIVIDUAL SYSTEM:

INVESTIGATIONS:

- Haemoglobin
- WBC count
- Platelet count
- Blood urea
- blood sugar
- Serum creatinine
- Serum Sodium
- Serum potassium
- HIC 1 @ 2
- Electrocardiograph
- Chest X-ray
- USG abdomen

ANNEXURE – II

ஒப்புதல் படிவம்

பெயர் :

பாலினம் :

வயது :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் பொது அறுவை சிகிச்சை பிரிவு மருத்துவ துறையில் பட்ட பயிலும் மாணவன் அவர்கள் மேற்கொள்ளும் **"PROSPECTIVE ANALYSIS OF FACTORS DECIDING SURGICAL INTERVENTION IN LIVER ABSCESS"** குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடன், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

ANNEXURE - III
MASTER CHART

S.No	name	age	sex	doa	ip no	character of pus		lobe involed		loculation		number oflesions		primary vs recurrent		recovered	dod	death
						thin	thick	right	left	uni	multi	single	multi	primary vs recurrent	recurrent			
1	renganathan	45	m	02-01-2018	188	no	yes	yes	no	no	yes	yes	no	yes	no	yes	09-01-2017	
2	sekar	60	m	06-01-2017	309	yes	no	no	yes	yes	no	yes	no	yes	no	yes	13-01-2017	
3	chellapan	52	m	09-01-2017	559	yes	no	no	yes	yes	no	no	yes	yes	no	yes	16-01-2017	
4	praveen	28	m	13-01-2017	980	no	yes	yes	no	no	yes	yes	no	yes	no	yes	21-01-2017	
5	sundaram	72	m	15-01-2017	1200	yes	no	no	yes	yes	no	no	yes	no	yes	yes	23-01-2017	
6	Palanisamy	50	m	18-01-2017	1800	yes	no	yes	no	no	yes	no	yes	yes	no	yes	21-01-2017	
7	elango	48	m	22-01-2017	2800	no	yes	no	yes	yes	no	yes	no	yes	no	yes	19-01-2017	
8	marimuthu	32	m	25-01-2017	3456	yes	no	yes	no	no	yes	yes	no	yes	no	yes	04-02-2017	
9	chellamal	71	f	28-01-2017	3896	no	yes	no	yes	yes	no	no	yes	yes	no	no		03-02-2017
10	muthusamy	55	m	29-01-2017	4653	yes	no	yes	no	yes	no	yes	no	yes	no	yes	08-02-2017	
11	ravi	45	m	30-01-2017	4809	no	yes	yes	no	no	yes	no	yes	yes	no	yes	10-02-2017	
12	suresh	43	m	02-02-2017	5538	yes	no	yes	no	yes	no	yes	no	yes	no	yes	11-02-2017	
13	vijay	53	m	04-02-2017	5045	yes	no	no	yes	yes	no	yes	no	no	yes	yes	11-02-2017	
14	uma	36	f	06-02-2017	7745	yes	no	yes	no	no	yes	yes	no	yes	no	yes	15-02-2007	
15	siva	49	m	09-02-2017	8777	no	yes	yes	no	yes	no	no	yes	yes	no	yes	18-02-2017	
16	devan	52	m	11-02-2017	9005	yes	no	no	yes	yes	no	yes	no	yes	no	yes	21-02-2017	
17	abraham	59	m	14-02-2017	10035	yes	no	yes	no	no	yes	yes	no	yes	no	yes	21-02-2017	
18	peter	52	m	17-02-2017	11098	no	yes	yes	no	yes	no	no	yes	yes	no	yes	25-02-2017	
19	Palanisamy	67	m	20-02-2017	11456	yes	no	no	yes	yes	no	yes	no	yes	no	yes	28-02-2017	
20	abdul	38	m	24-02-2017	11567	yes	no	yes	no	no	yes	yes	no	no	yes	yes	03-03-2017	
21	palniammal	56	f	26-02-2017	12090	no	yes	yes	no	yes	no	no	yes	yes	no	yes	05-03-2017	
22	palani	46	m	28-02-2017	12908	yes	no	yes	no	yes	no	yes	no	yes	no	yes	05-03-2017	
23	raju	44	m	02-03-2017	13876	yes	no	yes	no	no	yes	yes	no	yes	no	yes	06-03-2017	
24	kanan	49	m	04-03-2017	13976	yes	no	yes	no	yes	no	yes	no	yes	no	yes	11-03-2017	
25	perumal	56	m	06-03-2017	14196	yes	no	yes	no	yes	no	no	yes	yes	no	yes	13-03-2017	
26	soman	55	m	07-03-2017	14567	yes	no	no	yes	yes	no	yes	no	yes	no	yes	18-03-2017	
27	deepak	34	m	09-03-2017	14870	yes	no	yes	no	yes	no	yes	no	no	yes	yes	15-03-2017	
28	vijay	54	m	12-Mar	14987	no	yes	yes	no	yes	no	yes	no	yes	no	yes	21-03-2017	
29	dilip	58	m	14-03-2017	15004	yes	no	no	yes	no	yes	yes	no	yes	no	yes	22-03-2017	
30	sakthi	44	m	17-03-2017	15897	yes	no	yes	no	yes	no	yes	no	yes	no	yes	27-03-2017	
31	vignesh	51	m	19-03-2017	16440	no	yes	yes	no	yes	no	no	yes	yes	no	yes	01-04-2017	
32	radhika	38	f	22-03-2017	16509	no	yes	no	yes	no	yes	yes	no	yes	no	yes	01-04-2017	
33	rahamatulla	56	m	25-03-2017	16778	yes	no	yes	no	yes	no	yes	no	no	yes	yes	06-04-2017	
34	sathish	57	m	28-03-2017	16908	yes	no	yes	no	yes	no	yes	no	yes	no	yes	08-04-2017	
35	nagaraj	59	m	31-03-2017	17884	no	yes	no	yes	yes	no	no	yes	yes	no	yes	11-04-2017	
36	mohammed	48	m	03-04-2017	18456	yes	no	yes	no	no	yes	yes	no	yes	no	yes	14-04-2017	

S.No	name	age	sex	doa	ip no	character of pus		lobe involed		loculation		number oflesions		primary vs recurrent		recovered	dod	death
						thin	thick	right	left	uni	multi	single	multi	primary vs recurrent	recurrent			
37	iqbal	39	m	06-04-2017	18567	no	yes	no	yes	yes	no	yes	no	yes	no	yes	14-04-2017	
38	sharmila	65	m	08-04-2017	18976	yes	no	yes	no	yes	no	yes	no	yes	no	yes	16-04-2017	
39	sahruk	55	m	10-04-2017	19444	yes	no	yes	no	no	yes	no	yes	yes	no	yes	19-04-2017	
40	samabasivan	45	m	13-04-2017	19589	yes	no	yes	no	yes	no	yes	no	no	yes	yes	22-04-2017	
41	murgesan	40	m	16-04-2017	19987	yes	no	no	yes	yes	no	yes	no	yes	no	yes	25-04-2017	
42	vidya	44	f	18-04-2017	20055	no	yes	yes	no	no	yes	no	yes	yes	no	yes	28-04-2017	
43	prabhu	38	m	21-04-2017	20453	yes	no	no	yes	yes	no	yes	no	yes	no	yes	02-05-2017	
44	murugan	32	m	24-04-2017	20678	no	yes	yes	no	yes	no	yes	no	yes	no	yes	03-05-2017	
45	balan	41	m	26-04-2017	21457	yes	no	yes	no	no	yes	yes	no	yes	no	yes	05-05-2017	
46	balasakthi	54	m	29-04-2017	21598	yes	no	no	yes	yes	no	yes	no	no	yes	yes	08-05-2017	
47	chandran	60	m	30-04-2017	21765	no	yes	yes	no	yes	no	no	yes	yes	no	yes	11-05-2017	
48	samuel	53	m	02-05-2017	21890	no	yes	yes	no	no	yes	yes	no	yes	no	yes	14-05-2017	
49	jayakumar	33	m	06-05-2017	21991	yes	no	no	yes	yes	no	yes	no	yes	no	yes	14-05-2017	
50	nanjammal	39	f	07-05-2017	22538	yes	no	yes	no	yes	no	yes	no	yes	no	yes	15-05-2017	
51	vijayaraj	51	m	09-05-2017	22785	yes	no	no	yes	no	yes	yes	no	yes	no	yes	17-05-2017	
52	muthusamy	59	m	11-05-2017	22896	no	yes	yes	no	yes	no	no	yes	no	yes	yes	22-05-2017	
53	muthuraj	48	m	12-05-2017	22997	yes	no	yes	no	yes	no	yes	no	yes	no	yes	20-05-2017	
54	alwn	47	m	15-05-2017	23189	yes	no	no	yes	no	yes	yes	no	yes	no	yes	23-05-2017	
55	naveen	43	m	18-05-2017	23452	no	yes	no	yes	yes	no	no	yes	yes	no	yes	28-05-2017	
56	tajan	44	m	21-05-2017	23765	yes	no	yes	no	yes	no	yes	no	yes	no	yes	27-05-2017	
57	rajesh	54	m	24-05-2017	23878	no	yes	yes	no	no	yes	yes	no	yes	no	yes	02-06-2017	
58	dhanam	58	f	26-05-2017	24342	yes	no	yes	no	no	yes	yes	no	yes	no	yes	05-06-2017	
59	aswin	53	m	28-05-2017	24449	no	yes	no	yes	yes	no	yes	no	no	yes	yes	08-06-2017	
60	irfan	48	m	28-05-2017	24887	yes	no	yes	no	no	yes	yes	no	no	yes	yes	11-06-2017	
61	habbeb	45	m	30-05-2017	25004	no	yes	yes	no	yes	no	no	yes	yes	no	yes	14-06-2017	
62	jojin	30	m	02-06-2017	25223	yes	no	yes	no	yes	no	yes	no	yes	no	yes	15-06-2017	
63	mustafa	44	m	04-06-2017	25456	yes	no	no	yes	no	yes	yes	no	yes	no	yes	18-06-2017	
64	palanivel	56	m	05-06-2017	25785	yes	no	yes	no	yes	no	no	yes	yes	no	yes	19-06-2017	
65	manikar	57	m	08-06-2017	25841	yes	no	yes	no	yes	no	yes	no	yes	no	yes	19-06-2017	
66	saraswathi	47	f	10-06-2017	26006	no	yes	yes	no	no	yes	no	yes	no	yes	yes	22-06-2017	
67	manikandan	49	m	11-06-2017	26574	yes	no	yes	no	yes	no	yes	no	yes	no	yes	23-06-2007	
68	jei	50	m	13-06-2017	27689	yes	no	yes	no	yes	no	yes	no	yes	no	yes	22-06-2017	
69	vikram	51	m	15-06-2017	28999	no	yes	yes	no	no	yes	no	yes	yes	no	yes	24-06-2017	
70	rajallingam	63	m	17-06-2017	29754	yes	no	yes	no	yes	no	yes	no	yes	no	yes	27-06-2017	
71	sundaraja	64	m	18-06-2017	30187	yes	no	yes	no	yes	no	no	yes	yes	no	yes	28-06-2017	
72	lakshmi	68	f	21-06-2017	31457	no	yes	yes	no	no	yes	yes	no	no	yes	no		23-06-2007
73	kaliyappan	38	m	24-06-2017	31679	yes	no	yes	no	yes	no	yes	no	yes	no	yes	02-07-2017	
74	jyothi	35	m	27-06-2017	32409	yes	no	yes	no	yes	no	no	yes	yes	no	yes	03-07-2017	
75	vikas	45	m	28-06-2017	32786	yes	no	yes	no	no	yes	yes	no	yes	no	yes	05-07-2017	

S.No	name	age	sex	doa	ip no	character of pus		lobe involed		loculation		number oflesions		primary vs recurrent		recovered	dod	death
						thin	thick	right	left	uni	multi	single	multi	primary vs recurrent	recurrent			
76	panerselvan	32	m	30-06-2017	33564	no	yes	yes	no	yes	no	no	yes	yes	no	yes	08-07-2017	
77	saptagiri	56	m	01-07-2017	33976	yes	no	yes	no	yes	no	yes	no	no	yes	yes	10-07-2017	
78	moorthy	67	m	04-07-2017	34976	yes	no	no	yes	no	yes	yes	no	yes	no	yes	13-07-2017	
79	john	72	m	06-07-2017	35468	yes	no	yes	no	yes	no	no	yes	yes	no	no		08-07-2017
80	parvathi	78	f	07-07-2017	35897	yes	no	yes	no	yes	no	yes	no	yes	no	yes	14-07-2017	
81	dharan	56	m	09-07-2017	36887	no	yes	no	yes	no	yes	no	yes	yes	no	yes	16-07-2017	
82	alagesan	38	m	11-07-2017	37690	yes	no	no	yes	yes	no	yes	no	no	yes	yes	18-07-2017	
83	anil	58	m	14-07-2017	38965	yes	no	yes	no	yes	no	no	yes	yes	no	yes	21-07-2017	
84	palanal	53	f	16-07-2017	41269	no	yes	yes	no	no	yes	yes	no	yes	no	yes	24-07-2017	
85	remesh	59	m	19-07-2017	43678	no	yes	no	yes	yes	no	no	yes	yes	no	yes	28-07-2017	
86	murali	76	m	22-07-2017	44098	yes	no	yes	no	yes	no	yes	no	yes	no	no		22-07-2017
87	isaac	39	m	26-07-2017	45032	yes	no	yes	no	no	yes	no	yes	yes	no	yes	06-08-2017	
88	pallavi	47	f	27-07-2017	48765	yes	no	no	yes	yes	no	yes	no	no	yes	yes	08-08-2017	
89	ramalingam	44	m	29-07-2017	51008	yes	no	yes	no	yes	no	no	yes	yes	no	yes	10-08-2017	
90	eswanth	47	m	30-07-2017	55346	no	yes	no	yes	no	yes	yes	no	yes	no	yes	11-08-2017	
91	ganesh	40	m	31-07-2017	58678	no	yes	yes	no	yes	no	yes	no	yes	no	yes	13-08-2017	
92	palaniyyammal	78	f	03-08-2017	62986	yes	no	yes	no	yes	no	no	yes	yes	no	no		07-08-2017
93	jeyaraj	45	m	05-08-2017	64008	yes	no	no	yes	no	yes	yes	no	no	yes	yes	15-08-2017	
94	bijish	47	m	07-08-2017	68432	no	yes	yes	no	no	yes	yes	no	yes	no	yes	17-08-2017	
95	annamalai	67	m	10-08-2017	72007	no	yes	yes	no	yes	no	no	yes	no	yes	yes	18-08-2017	
96	jaya	37	f	12-08-2017	75321	yes	no	no	yes	no	yes	no	yes	yes	no	yes	22-08-2017	
97	vadivel	49	m	14-08-2017	84098	no	yes	yes	no	yes	no	yes	no	yes	no	yes	24-08-2017	
98	hassan	53	m	17-08-2017	88543	yes	no	yes	no	yes	no	no	yes	yes	no	yes	26-08-2017	
99	suresh	55	m	20-08-2017	96006	no	yes	no	yes	no	yes	yes	no	yes	no	yes	30-08-2017	
100	jayanthi	59	f	22-08-2017	100060	yes	no	yes	no	yes	no	no	yes	yes	no	yes	04-09-2017	
101	mayandi	72	m	26-08-2017	100456	no	yes	yes	no	no	yes	yes	no	yes	no	yes	06-09-2017	
102	joseph	37	m	28-08-2017	102678	yes	no	no	yes	yes	no	yes	no	yes	no	yes	09-09-2017	
103	sharmila	49	f	30-08-2017	103456	yes	no	yes	no	yes	no	no	yes	yes	no	yes	10-09-2017	
104	marimuthu	45	m	03-09-2017	104876	no	yes	yes	no	no	yes	yes	no	no	yes	yes	12-09-2017	
105	surya	43	m	05-09-2017	107654	yes	no	yes	no	yes	no	no	yes	yes	no	yes	15-09-2017	
106	elamaran	49	m	06-09-2017	116789	yes	no	no	yes	yes	no	yes	no	yes	no	yes	17-09-2017	
107	nanjappan	58	m	08-09-2017	118907	no	yes	no	yes	no	yes	no	yes	yes	no	yes	20-09-2017	
108	karuppusamy	68	m	10-09-2017	135678	yes	no	no	no	no	yes	yes	no	yes	no	yes	20-09-2017	
109	beegum	30	f	11-09-2017	148906	no	yes	yes	no	yes	no	yes	no	yes	no	yes	23-09-2017	
110	sheik	29	m	13-09-2017	154328	yes	no	yes	no	no	yes	yes	no	yes	no	yes	22-09-2017	
111	palanisamu	73	m	16-09-2017	167895	yes	no	yes	no	yes	no	no	yes	yes	no	no		18-09-2017
112	kanagaraj	45	m	18-09-2017	174789	yes	no	no	yes	yes	no	no	yes	no	yes	yes	25-09-2017	
113	ganesh	38	m	21-09-2017	176542	yes	no	yes	no	no	yes	yes	no	yes	no	yes	28-09-2017	
114	kuppusamy	79	m	23-09-2017	187609	no	yes	yes	no	yes	no	yes	no	yes	no	yes	01-10-2017	

S.No	name	age	sex	doa	ip no	character of pus		lobe involed		loculation		number oflesions		primary vs recurrent		recovered	dod	death
						thin	thick	right	left	uni	multi	single	multi	primary vs recurrent	recurrent			
115	kuppammal	63	f	25-09-2017	187650	yes	no	yes	no	yes	no	yes	no	yes	no	yes	02-10-2017	
116	karuppan	68	m	28-09-2017	188954	no	yes	no	yes	no	yes	no	yes	no	yes	yes	05-10-2017	
117	kanan	42	m	30-09-2017	198753	yes	no	yes	no	yes	no	yes	no	yes	no	yes	07-10-2017	
118	vinth	41	m	01-10-2017	199976	yes	no	no	yes	no	yes	yes	no	yes	no	yes	10-10-2017	
119	sivakumar	34	m	03-10-2017	200078	yes	no	yes	no	yes	no	no	yes	yes	no	yes	11-10-2017	
120	kaliyappan	76	m	06-10-2017	200367	yes	no	yes	no	no	yes	yes	no	yes	no	yes	22-10-2017	
121	john	48	m	09-10-2017	201654	no	yes	yes	no	yes	no	no	yes	yes	no	yes	15-10-2017	
122	dhanalakshmi	48	f	12-10-2017	207896	yes	no	no	yes	yes	no	yes	no	no	yes	yes	17-10-2017	
123	dinakar	56	m	15-10-2017	209965	yes	no	yes	no	no	yes	yes	no	yes	no	yes	24-10-2017	
124	velayudham	60	m	19-10-2017	215678	no	yes	yes	no	yes	no	no	yes	yes	no	yes	27-10-2017	
125	vivek	38	m	22-10-2017	217890	yes	no	yes	no	yes	no	yes	no	yes	no	yes	31-10-2017	
126	ananth	48	m	26-10-2017	223567	yes	no	yes	no	no	yes	no	yes	yes	no	yes	31-10-2017	
127	prasnth	33	m	02-11-2017	224465	no	yes	no	yes	yes	no	yes	no	yes	no	yes	09-11-2017	
128	mohan	46	m	05-11-2017	225680	yes	no	yes	no	no	yes	yes	no	yes	no	yes	14-11-2017	
129	kumar	25	m	07-11-2017	226785	no	yes	yes	no	yes	no	no	yes	no	yes	yes	17-11-2017	
130	uday	49	m	10-11-2017	236643	yes	no	yes	no	yes	no	yes	no	yes	no	yes	18-11-2017	
131	tamilselvan	55	m	15-11-2017	237680	yes	no	no	yes	yes	no	yes	no	yes	no	yes	23-11-2017	
132	kamaraj	53	m	19-11-2017	238765	no	yes	yes	no	no	yes	yes	no	yes	no	yes	28-11-2017	
133	rangammal	33	f	22-11-2017	240005	yes	no	no	yes	yes	no	no	yes	no	yes	yes	29-11-2017	
134	gowtham	29	m	26-11-2017	241278	no	yes	yes	no	yes	no	yes	no	yes	no	yes	04-12-2017	
135	narayanan	55	m	28-11-2017	243789	yes	no	yes	no	no	yes	yes	no	yes	no	yes	08-12-2017	
136	ojinbasha	34	m	30-11-2017	244098	yes	no	yes	no	yes	no	no	yes	no	yes	yes	10-12-2017	
137	surya	45	m	06-12-2017	245673	yes	no	no	yes	yes	no	yes	no	yes	no	yes	13-12-2017	
138	ajith	28	m	11-12-2017	249865	no	yes	yes	no	no	yes	yes	no	yes	no	yes	19-12-2017	
139	mary	35	f	14-12-2017	254764	yes	no	yes	no	yes	no	yes	no	yes	no	yes	22-12-2017	
140	thirumoorthy	37	m	17-12-2017	255632	yes	no	no	yes	yes	no	no	yes	yes	no	yes	22-12-2017	
141	arun	49	m	20-12-2017	256789	yes	no	yes	no	no	yes	yes	no	yes	no	yes	26-12-2017	
142	ashok	36	m	22-12-2017	257894	no	yes	yes	no	yes	no	yes	no	yes	no	yes	30-12-2017	
143	avinash	45	m	24-12-2017	258790	yes	no	no	yes	no	yes	yes	no	no	yes	yes	05-01-2018	
144	ajay	39	m	28-12-2017	259933	yes	no	yes	no	yes	no	no	yes	yes	no	yes	08-01-2018	